POSTGRADUATE LECTURE COURSE — BEGINNING MARCH 3

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James A Kleeman

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In the Early Recognition of Protein Deficiency

Unsupervised dietary curtailment and self-imposed food restrictions, not infrequently observed in elderly patients and in those desirous of preventing weight gain or losing weight, are apt to lead to multiple nutritional derangements. Not the least important among these, and often overlooked, is protein deficiency.

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THE ROLE OF LANCEFIELD GROUPS OF BETA-HEMOLYTIC STREPTOCOCCI IN RESPIRATORY INFECTIONS*

THE COMMISSION ON ACUTE RESPIRATORY DISEASES†

IT IS widely recognized that Lancefield Group A streptococci are the causal agents in the majority of streptococcal infections in man. In suppurative or generalized infections the role of other groups of streptococci has been established by the isolation of these organisms in pure culture from collections of pus or from the blood stream. In respiratory infections the problem is more difficult, since various Lancefield groups of beta-hemolytic streptococci are found in the oropharynx of normal subjects. Thus, the isolation of one of these groups from the throat of a patient with respiratory disease does not necessarily establish an etiologic relation. Other methods are therefore required to ascertain the relation of the beta-hemolytic streptococcus to the pathologic process.

Previous reports from this laboratory have emphasized the value of serologic tests in the diagnosis of streptococcal respiratory infections. Studies of a food-borne outbreak showed that at least 85 per cent of patients with known streptococcal disease develop either antistreptolysin or antifibrinolysin during the convalescent period[1] and that these antibodies rarely develop in patients who do not exhibit other evidence of streptococcal infection.[2]

In serologically proved cases of pharyngitis and tonsillitis caused by beta-hemolytic streptococci, certain clinical, bacteriologic and epidemiologic findings have been of value in specific diagnosis. The clinical characteristics include a history of a sudden onset of sore throat, with associated constitutional symptoms, the physical signs of confluent exudate on the tonsils or pharynx, diffuse injection and edema of the oropharyngeal mucous membranes and enlargement and tenderness of the cervical lymph nodes.

Laboratory studies usually show a polymorphonuclear leukocytosis in such streptococcal infections. Likewise, single or multiple cultures of the oropharynx ordinarily show large numbers of beta-hemolytic streptococci.[3] Not infrequently, however, such clinical and bacteriologic findings are misleading, for many patients, with or without exudative tonsillitis or pharyngitis but harboring beta-hemolytic streptococci in the oropharynx, fail to develop specific antibodies during convalescence.[3, 4]

Finally, certain epidemiologic factors have been found to be of value in determining the role of beta-hemolytic streptococci in respiratory disease. It has sometimes been possible to demonstrate a concentration of cases harboring the same group or type of organism.[5] Cultural surveys in the normal population and in patients with respiratory disease have been particularly valuable. In one study the total incidence and distribution of groups of beta-hemolytic streptococci were identical in normal soldiers and in patients with respiratory disease who did not develop specific antibodies during convalescence.[4] It was therefore concluded that the majority of patients harboring beta-hemolytic streptococci and failing to develop antibodies were merely carriers of the organisms who had contracted a respiratory infection caused by other agents.

The present study of 3026 cases of respiratory disease was undertaken to determine the role of various groups of streptococci in ordinary respiratory infections. Many of the patients were found to harbor beta-hemolytic streptococci in the oropharynx. Not only Group A organisms but also streptococci of groups B, C, F, G, H and L were encountered. By the use of bacteriologic, epidemiologic, clinical and serologic methods it was shown that infections were caused by Lancefield groups A, C and G and probably also by groups B and F. The various features of the illnesses produced by the different groups of streptococci are described below.

*From the Respiratory Diseases Commission Laboratory (Regional Station Hospital Section 2, Fort Bragg, North Carolina (now located in the Department of Preventive Medicine, Western Reserve University School of Medicine, Cleveland 6, Ohio).

This investigation was aided by the support of the Commission on Acute Respiratory Diseases, Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army, Preventive Medicine Service, Office of the Surgeon General, United States Army, and by grants from the Commonwealth Fund, the W. K. Kellogg Foundation, the John and Mary R. Markle Foundation and the International Health Division of the Rockefeller Foundation to the Board for the Investigation and Control of Influenza and Other Epidemic Diseases for the Commission on Acute Respiratory Diseases.

†Members and professional associates of the Commission on Acute Respiratory Diseases are Lieutenant Colonel John H. Dingle, M.C., A.U.S., director; Lieutenant Colonel Theodore J. Abernethy, M.C., A.U.S.; Major George F Badger, M.C., A.U.S.; Major Norman L. Cressy, M.C., A.U.S.; A. E. Feller, M.D.; Irving Gordon, M.D.; Major Alexander D. Langmuir, M.C., A.U.S.; Charles H. Rammelkamp, Jr., M.D.; Major Elias Strauss, M.C., A.U.S., and Captain Hugh Tatlock, M.C., A.U.S.

Materials and Methods

Source of Cases

The studies were carried out in the Field Artillery Replacement Training Center at Fort Bragg. The soldiers in this organization arrived in large groups from reception centers located throughout the country. At the time of arrival the majority of the men had been in the Army for less than ten days. For a period of eight to seventeen weeks they received basic training and were then transferred to other stations.

The epidemiologic and clinical features of the respiratory infections occurring in this organization will be described in detail elsewhere.[5] Briefly, each group of recruits observed during the winter months experienced an epidemic of respiratory infections that resulted in the hospitalization of 15 to 30 per cent of the entire organization.* Although the majority of these illnesses were acute, undifferentiated respiratory diseases,[5] other infections, such as streptococcal tonsillitis and pharyngitis, nonstreptococcal exudative pharyngitis or tonsillitis, influenza A and B, bacterial pneumonia and primary atypical pneumonia, were encountered. In the present study there were few specific contagious diseases.

Between 1943 and 1945 a total of 3026 patients with respiratory infections admitted to the hospital were studied. This included all patients with respiratory infections entering the hospital from selected organizations of recruits during the winter and spring of 1943-1944 and 1944-1945 and 116 consecutive cases of exudative tonsillitis or pharyngitis observed between April and June, 1943.[3] The patients were examined soon after admission, and cultures from the throat were obtained. Usually, a second and a third culture were taken from patients who exhibited exudative tonsillitis or pharyngitis or who harbored beta-hemolytic streptococci in the throat as determined by the first culture. An initial sample of blood was drawn at the time of hospitalization, and in 92 per cent of cases, one or more convalescent specimens were obtained three to eight weeks later.

Carrier Surveys

Throughout most of the period of this study carrier studies were carried out in large samples of recruits who were on active duty. These cultural surveys were made between January and May, 1944 and 1945, during which time most of the patients with respiratory infections were studied.

Laboratory Procedures

The throat cultures were obtained by the swabbing of both tonsils or tonsillar fossas and the posterior pharynx. The swab was either streaked directly on a blood-agar plate† or placed in 3 to 5 cc. of tryptose phosphate broth, and after thorough mixing, 0.1 cc. was transferred to a blood-agar plate. The plates were then incubated at 37°C. for twenty-four hours, following which they were examined for colonies showing beta hemolysis under a wide-field binocular microscope. In general, only one colony was picked for grouping and typing, unless two distinct types were recognized. The grouping of the beta-hemolytic streptococci was accomplished by the capillary tube-precipitin method, extracts prepared by Fuller's technic[6] being used. Commercial serums were available for testing groups A, B, C, D, E, F, G, H and L.

The streptococcal antibodies employed in serologic diagnosis were antistreptolysin and antifibrinolysin. Antistreptolysin O titers were determined by a modification of the method of Hodge and Swift.[7] The technic used to measure antifibrinolysin has been described elsewhere.[8] The fibrinolysin employed routinely in this test was prepared from a Group A beta-hemolytic streptococcus (No. 98); in a number of cases, however, determinations were also made with streptococci of groups C and G as the source of fibrinolysin. In both tests for streptococcal antibodies the acute and convalescent specimens of serum were examined at the same time. Under these conditions an increase of titer of either antibody of two or more dilution increments during the convalescent period has been shown to be highly indicative of a specific reaction to the streptococcus.[2] In the present study, therefore, only changes of this magnitude demonstrated within a period of eight weeks — usually within three weeks — were considered significant.

Cultural Surveys

The frequency with which the various Lancefield groups of streptococci were found in the oropharynx of normal soldiers and in patients with respiratory infections admitted to the hospital is shown in Table 1. The carrier rates for all beta-hemolytic streptococci in the normal soldiers varied between 8.0 and 9.7 per cent, averaging 8.9 per cent. The rate among patients hospitalized for respiratory disease was 13.9 per cent.

The 5 per cent difference between these rates is largely accounted for by the fact that Group A streptococci were isolated more frequently in the soldiers with respiratory infections than in healthy soldiers. This organism was obtained in the former in 9.5 per cent, and in the latter, in 5.4 per cent — a difference of 4.1 per cent. These data suggest that Group A streptococci were responsible for the majority of the streptococcal infections.

Lancefield groups B, C, F and G were isolated from the oropharynx of both healthy subjects and hospitalized patients, whereas streptococci of groups D, E and H were not found. In the hospitalized soldiers, a Group L streptococcus was isolated from 1 patient at the time of admission. With the excep-

*A soldier with respiratory disease was hospitalized if the oral temperature was over 100°F.

†All blood-agar plates contained 0.005 per cent para-aminobenzoic acid.

tion of Group A, there were no significant differences in the carrier rates of these various Lancefield groups between the normal soldiers and those hospitalized for respiratory disease. These data indicate that the organisms did not play an important role in the respiratory infections studied. It cannot be concluded, however, that no infections were caused by these streptococci.

Serologic Studies

As pointed out above, the isolation of beta-hemolytic streptococci from the throats of patients with respiratory disease does not necessarily indicate a causal relation, and this point is emphasized by the fact that the same organisms were recovered from normal soldiers. The number of streptococcal infections occurring in the hospitalized soldiers may be estimated by differences between the carrier rates in the normal and hospitalized soldiers; such studies, however, do not disclose the person who is infected with the organism. It is necessary, therefore, to use other methods to establish the validity of a diagnosis of a streptococcal respiratory infection in the individual patient.

A serologic diagnosis of streptococcal infection was accomplished by the demonstration of an increase in the antistreptolysin or antifibrinolysin titer of the serum obtained during convalescence. Streptolysin O is produced by organisms belonging to Lancefield groups A, C, and G,[9] and fibrinolysin is produced principally by the same groups, although a few strains of other groups have been observed to cause lysis of fibrin clots.[10] Thus, these two antibody tests are at present considered to be of value only in the diagnosis of infections caused by streptococci of groups A, C and G.

To evaluate the results of the antibody determinations in the hospitalized patients, it was first necessary to establish the frequency with which an increase in titers of antistreptolysin or antifibrinolysin occurred in the normal population. For this purpose a serum specimen was obtained from soldiers on active duty, following which the oropharynx was cultured at weekly intervals. A second specimen of blood was taken three to eight weeks later. Antibody determinations on these sets of serum disclosed that of 236 soldiers who did not harbor beta-hemolytic streptococci, none developed antibodies.[2] There were 45 subjects who harbored Group A organisms without illness, 1 of whom developed antibodies. In sets of serum collected from 37 soldiers harboring Group C streptococci at some time during the three to eight weeks of study, an increase in the antistreptolysin titer was demonstrated in 1 case. This soldier did not enter the hospital, nor was he seen in the local dispensary. Three of four cultures of the throat taken between the two samples of blood showed Group C streptococci. There was no significant change in antibody titer in sets of serum collected from 35 soldiers harboring Group G streptococci.

Table 1. *Comparison of Carrier Rates of Various Lancefield Groups of Beta-Hemolytic Streptococci in 3853 Normal Soldiers and 3026 Hospitalized Soldiers with Respiratory Infections.*

Organism	Rates in Normal Soldiers				Rates in Hospitalized Soldiers*
	March-May, 1944	January, 1945	March, 1945	1944-1945 combined	1943-1945
	%	%	%	%	%
Group A	5.2	4.6	6.7	5.4	9.5
Group B	—	0.2	0.3	0.1	0.2
Group C	1.0	1.9	1.5	1.4	1.8
Group F	0.1	0.2	0.2	0.2	0.1
Group G	1.6	2.3	0.7	1.6	1.4
Group L	—	—	—	—	0.1
Combination	—	—	—	—	0.1
Not grouped	0.1	0.2	0.3	0.2	0.7
Total carrier rate	8.0	9.4	9.7	8.9	13.9
Number of cultures taken	1585	1229	1039	3853	3026

*Admission cultures.

These results demonstrated that in the population studied streptococcal antistreptolysin and antifibrinolysin did not develop in the absence of beta-hemolytic streptococci; moreover, such antibodies were rarely demonstrated in the serum of normal soldiers who harbored these organisms.

Serums taken during the acute and convalescent phases were available for study from 429 — 92 per cent — of the 465 hospitalized men who harbored beta-hemolytic streptococci in the oropharynx in any of three throat cultures. Of the 429 patients, 161, or 37.4 per cent, developed specific antibodies during the period of convalescence (Table 2). The majority of the increases in antibodies were observed in serums collected from patients harboring Group A streptococci. Of the sets of serum from patients with Group A streptococci alone, approximately half exhibited an increased titer during convalescence, and 39 per cent of patients with Group A in combination with some other group developed antibodies. Of 43 patients with Group C and 41 with Group G streptococci, 16 and 12 per cent, respectively, developed antibodies during the convalescent period. One patient from whom Group L was isolated developed antibodies.

Since the demonstration of an increase of antistreptolysin or antifibrinolysin titer during early convalescence is a highly specific test[2] and since such increases were rarely observed in the normal populations studied, it is concluded that the demonstration of a significant increase in these antibodies in the 161 hospitalized soldiers indicated that the respiratory infections were caused by beta-hemolytic streptococci.

TABLE 2. *Antibody Response in Patients with Respiratory Infections Harboring the Beta-Hemolytic Streptococcus.*

ORGANISM	NO. OF PATIENTS WITH ISOLATION IN ANY OF THREE CULTURES	NO. OF PATIENTS TESTED FOR ANTIBODY RESPONSE	ANTIBODY RESPONSE*	
			NO.	PERCENTAGE
Group A	301	283	139	49.1
Group B	6	5	0	
Group C	47	43	7	15.9
Group F	11	11	0	
Group G	44	41	5	12.1
Group H	1	1	0	
Group L	1	1	1	
Not grouped	33	24	2	8.3
Combinations:				
Group A and others	19	18	7	38.8
Others	2	2	0	0
Totals	465	429	161	
Average				37.4

*Either antistreptolysin or antifibrinolysin.

The frequency distribution of the Lancefield groups of streptococci in the 161 serologically proved cases of endemic streptococcal respiratory infections is presented in Table 3. Group A organisms alone were isolated from 86 per cent, and Group A alone or in combination with other groups accounted for 90 per cent. The remaining 10 per cent of cases occurred in patients harboring other groups, the largest being Group C with 7 patients, or 4 per cent of this total. Group G was isolated in 3 per cent of the infections.

TABLE 3. *Lancefield Groups of Beta-Hemolytic Streptococci Isolated from Throat Cultures of 161 Patients with Respiratory Disease Who Developed Specific Antibodies During Convalescence.*

ORGANISM	TOTAL NO. OF PATIENTS	PERCENTAGE OF TOTAL
Group A	139	86
Group A with other groups	7	4
Group C	7	4
Group G	5	3
Not grouped and others	3	2

EPIDEMIOLOGIC, CLINICAL, BACTERIOLOGIC AND IMMUNOLOGIC STUDIES

A detailed study was made of the cases of respiratory disease in which beta-hemolytic streptococci were recovered from the oropharynx. The purpose of this investigation was not only to establish the relation of the different Lancefield groups to the individual infection but also to describe the response of the host to such infections.

Group A

The difference in the carrier rates of Group A beta-hemolytic streptococci in the normal soldiers and the patients hospitalized for respiratory disease indicated that 124 (4.1 per cent) of the 3026 cases had been caused by this organism. Serologic studies showed that 139 patients had an infection due to Group A streptococci as determined by the demonstration of an increase in the antistreptolysin or antifibrinolysin titer during convalescence.

The serologically proved infections occurred essentially in a sporadic fashion; only a few small, localized outbreaks caused by a single type of Group A streptococcus were recognized.

Among the 139 patients who harbored Group A streptococci and developed antibodies 101, or 73 per cent, had exudative tonsillitis or pharyngitis. By contrast, the incidence of exudative lesions of the throat in the patients who harbored Group A organisms but failed to develop antibodies was only 34 per cent.

Clinical and laboratory data obtained on patients with exudative tonsillitis and pharyngitis due to Group A streptococci have been presented elsewhere in detail.[3] These patients usually complained of soreness of the throat and such constitutional symptoms as feverishness, chilliness, headache and anorexia. Inspection of the throat showed that the exudate was usually discrete to confluent and that the mucous membranes were diffusely injected and somewhat edematous. The regional lymph nodes were likely to be enlarged and tender. The average white-cell count was 16,000, and cultures of the throat showed that Group A streptococci predominated in approximately half the patients. The course of the illness was short, the symptoms usually disappearing by the fourth or fifth day.

The patients who did not have exudative lesions in the throat were seldom thought to have had streptococcal infections when examined at the time of admission. A diagnosis of the specific nature of the illness was possible only after bacteriologic and serologic studies had been completed.

Group B

Six patients were found to harbor Group B streptococci. As expected, an increase in antistreptolysin or antifibrinolysin titer during convalescence was not demonstrated in the serum of the 5 patients tested, since Group B streptococci do not produce streptolysin or fibrinolysin.

There was no evidence of focal concentration of the cases, which occurred sporadically throughout the three-year period. Three of the patients were thought to have undifferentiated acute respiratory

disease.[5] The remaining 3 patients had exudative tonsillitis. In 1 case all three cultures yielded Group B streptococci, and in one culture this organism accounted for more than 50 per cent of the colonies. The total white-cell count in this patient was 13,600. The second patient had discrete areas of exudate on both tonsils, as well as considerable edema of the soft palate. The lymph nodes were not large or tender. Only one of three cultures showed Group B streptococci, and the white-cell count was 14,200. The third patient with exudative tonsillitis showed diffuse redness and swelling around one tonsil, so that a diagnosis of peritonsillitis was made. The regional lymph nodes were enlarged and tender. The white-cell count was only 8600. A course of penicillin therapy resulted in recovery without abscess formation.

The clinical evidence thus suggested that 3 patients had a respiratory infection caused by the Group B streptococcus. These patients exhibited exudative lesions on the tonsils; 1 developed peritonsillitis, 1 showed enlarged and tender lymph nodes and in 2 the total white-cell count was elevated.

Group C

The carrier rate for Group C streptococci in the normal and in the hospitalized soldiers was practically identical, being 1.4 and 1.8 per cent respectively. These figures indicated that the majority of the patients were merely carriers and did not have a true streptococcal illness. This was confirmed by the serologic studies, which showed that only 7 hospitalized subjects harboring Group C streptococci developed antibodies during convalescence (Tables 2 and 4).

The illnesses in these 7 patients occurred sporadically throughout the entire period of study. At the time of admission to the hospital, 6 patients complained of a sore throat. In 3 cases inspection showed areas of discrete exudate on the tonsils. The maximum temperature recorded during hospitalization varied from 99.4 to 103.4°F., and the course of illness was similar to that of a mild streptococcal infection.[3] In the majority of cases the temperature became normal within seventy-two hours.

The laboratory studies showed that the average total white-cell count was 10,735. In the 3 patients who had exudative lesions on the tonsils, the total white-cell counts varied between 11,150 and 15,600. Two or more cultures were obtained from the throat in all cases during the first three days of hospitalization. In 6 cases the initial culture showed Group C streptococci. In 5 the organisms were isolated on two successive days. In 2 subjects Group C streptococci accounted for over 50 per cent of the colonies on the blood agar plate.

The serums of all 7 patients showed a significant increase in antistreptolysin titer during the convalescent period, but in only 1 subject (Case T-101) did the antifibrinolysin titer increase significantly. Several of the sets of serum (Cases T-101, O-258, O-522, O-581 and O-1070) were tested with fibrinolysin obtained from Group C as well as from Group A streptococci, and no essential difference was observed in the antibody titer. An increase in streptococcal antibodies was demonstrated within twenty-seven days following admission to the hospital in 6 patients. Thus, the clinical and laboratory studies, as well as the bacteriologic and serologic findings, indicated that the majority of the 7 cases presented in Table 4 represented infection by Group C streptococci.

The temperature chart of one of these patients (Case O-581) is presented in Figure 1. This eighteen-

TABLE 4. *Clinical and Laboratory Data on 7 Patients with Respiratory Disease Who Harbored Group C Streptococci and Developed Streptococcal Antibodies.*

Case No.	Date of Hospital Admission	Age	Sore Throat	Exudate	Maximum Temperature	White-cell Count	Cultures for Group C Streptococci			Interval after Admission	Anti-streptolysin Titer	Anti-fibrinolysin Titer
							First	Second	Third			
		yr.			*°F.*					*days*	*units*	*units*
T-101	5/20/43	20	+	+	102	11,400	+	+	0	1	50	<25
										23	159	36
T-120	6/ 1/43	19	+	+	103	11,150	0	0	+	1	100	56
										39	625	56
E-292	3/28/44	18	+	0	99.4	8,350	+	++	++	1	200	<25
										24	317	—
										42	500	<25
O-258	1/ 7/45	24	0	0	103.4	12,200	+	+		1	250	56
										16	500	56
										30	500	—
										51	400	56
O-522	1/23/45	27	+	0	102.2	9,050	+	+		2	125	36
										17	250	56
										25	317	36
O-581	1/26/45	18	+	+	103	15,600	++	++		1	200	<25
										27	400	<25
										48	317	<25
O-1070	3/10/45	30	+	0	100.4	7,400	+	0		1	125	<25
										8	200	<25

year-old soldier developed a nonproductive cough and mild coryza a week prior to admission to the hospital. Five days later he noticed a sore throat, as well as slight chilliness without a true rigor.

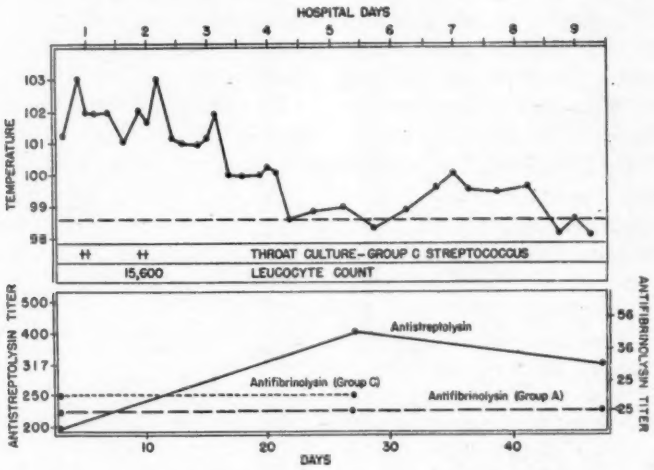

FIGURE 1. *Clinical Chart of a Patient with a Group C Streptococcus Infection.*

Inspection of the throat showed that the tonsils and surrounding tissues were diffusely injected, with areas of discrete exudate on both tonsils. The cervical lymph nodes were large and tender. The white-cell count on the second hospital day was 15,600, with 69 per cent neutrophils. The antistreptolysin titer increased from 200 to 400 units in twenty-six days, but no increase in antifibrinolysin was demonstrated in tests made from either Group A or Group C streptococci.

Group F

Eleven patients with respiratory illnesses were found to harbor Group F streptococci in the throat, and all these patients entered the hospital in March and April, 1944, when 857 patients admitted to the hospital were studied; there was no other evidence that these cases occurred in an epidemic fashion, since all the soldiers were housed in different barracks. A survey of the carrier rate for beta-hemolytic streptococci made during the same period showed Group F organisms in only 2, or 0.12 per cent, of 1568 soldiers on active duty. On the basis of the first hospital culture, 3, or 0.35 per cent, of 857 patients harbored the organism.

In the hospital 6 patients were housed in one ward, and 4 in another. This suggested the possibility of cross infection in the hospital; examination of the dates of admission and discharge, however, revealed that cross infections were unlikely in the majority of cases.

There was some clinical evidence that a few of the patients with Group F streptococci had a streptococcal illness. Seven patients* were found to have exudative tonsillitis or pharyngitis. The temperature curves, throat cultures and white-cell counts are presented in Figure 2. Three patients (Cases E-390, E-426 and E-531) also showed enlarged and tender cervical lymph nodes. Several showed a rather brisk febrile response.

In only 2 patients were Group F streptococci isolated in all three cultures; in 3 others, two cul-

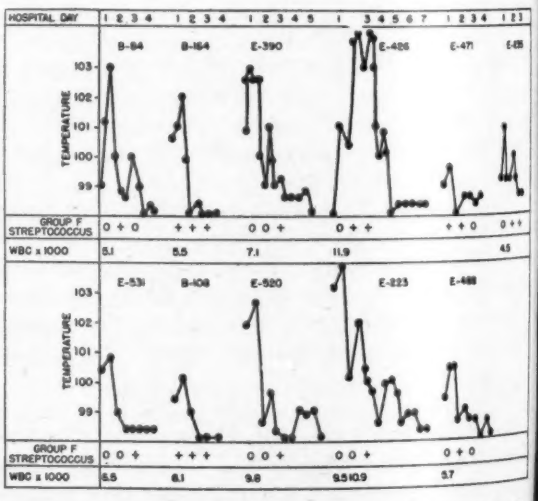

FIGURE 2. *Temperature Charts of Eleven Patients with Respiratory Infections Who Harbored Group F Streptococci in the Oropharynx.*

tures showed this organism. The white-cell count was slightly elevated in only 2 patients (Cases E-426 and E-223).

*Cases B-84, B-164, E-390, E-426, E-471, E-235 and E-531.

Group G

The average carrier rate for this group of streptococci was 1.6 per cent in the normal soldiers and 1.4 per cent in the patients with respiratory disease. It is thus apparent that few infections were caused by this organism. Serologic tests showed that 5 patients harboring Group G streptococci developed antibodies during convalescence. A summary of the data in these cases is presented in Tables 2 and 5. All but 1 of the patients complained of a mild sore throat at the time of the first interview, and in 1 case exudate was present on the tonsils. The maximum oral temperatures observed during the course of these illnesses varied from 100.8 to 103°F. The temperature became normal within two days in 4 cases, and in 1 it reached a normal level within three days.

TABLE 5. *Clinical and Laboratory Data on 5 Patients with Respiratory Disease Who Harbored Group G Streptococci and Developed Streptococcal Antibodies.*

Case No.	Date of Hospital Admission	Age	Sore Throat	Exudate	Maximum Temperature	White-cell Count	Cultures for Group G Streptococci			Interval after Admission	Anti-streptolysin Titer	Anti-fibrinolysin Titer
							First	Second	Third			
		yr.			*°F.*					*days*	*units*	*units*
B-8	2/13/44	28	+	0	102	9,400	+			1	125	< 25
										10	159	—
										27	200	<25
										32	200	—
E-549	4/23/44	35	0	0	101.8	9,200	0	+	0	1	50	<25
										19	200	25
										33	159	—
O-6	10/21/44	23	+	+	103	—	+	+	+	2	125	<25
										25	250	25
										66	400	25
										79	400	25
										95	400	25
O-694	1/31/45	18	+	0	102.4	8,650	+			1	63	25
										13	100	36
										24	100	36
										32	83	36
O-971	2/27/45	18	+	0	100.8	5,750	+	+	0	2	250	125
										32	500	125

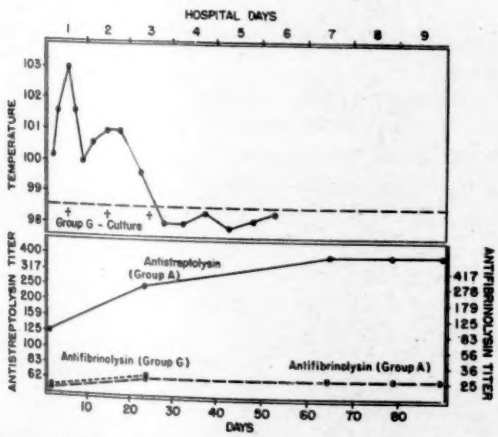

FIGURE 3. *Clinical Chart of a Patient with a Group G Streptococcus Infection.*

The temperature chart and the serologic studies of 1 patient (Case O-6) are shown in Figure 3. The onset of this patient's illness was twenty-four hours prior to admission to the hospital, when a sore throat, malaise, chilliness and a slight cough developed. Examination showed diffusely injected and swollen tonsils on which areas of discrete exudate were present. The cervical lymph nodes were small and not especially tender. Cultures on three successive days showed Group G streptococci. The antistreptolysin titer on admission was 125, and on the twenty-fifth day was 250. Antifibrinolysin determinations were made with the use of both Group A and Group G streptococci as the source of fibrinolysin. The titer with both fibrinolysins, which had been less than 25 units on the initial serum specimen, had increased to only 25 units on the twenty-fifth day.

Case O-694 is of interest since the increase in antistreptolysin titer was demonstrated on the thirteenth day of illness. The titer of serum specimens obtained twelve and nine days before admission was 63 units of antistreptolysin. Also, cultures of the throat obtained thirteen, twelve and one days prior to admission showed Group G streptococci. It therefore appears that the increase in antistreptolysin titer was due to this organism. No increase in titer to fibrinolysins of groups G, C and A was demonstrated in the serums obtained from this patient.

The first two cultures in Case O-971 showed Group G streptococci, and the antistreptolysin titer increased from 250 to 500 units in thirty days. Previous specimens of blood obtained fifteen and fifty-seven days prior to admission to the hospital showed a titer of 250 units. There was no change in the antifibrinolysin titer when tested by fibrino-

lysins prepared from Group A, Group C and Group G streptococci.

In Case E-549 only one culture was positive for Group G streptococci, but the antistreptolysin titer increased within nineteen days after admission to the hospital. Case B-8 also had only one culture that showed a Group G streptococcus. This patient entered the hospital because of weakness, chilliness and feverishness of two days' duration. The only respiratory symptoms were sneezing, coryza and a slight cough. Seven days after entry, the patient was discharged from the hospital, only to return two days later, when a blood specimen showed that the antistreptolysin titer had increased from 125 to 159 units. Cultures of the throat and sputum again showed Group G streptococci. A throat culture taken twelve days after the first admission showed a Group A, nontypable streptococcus. It seems probable, although not definite, that the illness in this patient was due to Group G, since an antibody response had occurred prior to the isolation of the Group A organism.

Thus, 5 of the 41 patients with respiratory disease who harbored Group G streptococci in the oropharynx exhibited serologic evidence of a streptococcal infection (Table 5). It is of some interest that only 1 of these patients had exudative tonsillitis. The white-cell count was not elevated in the 4 patients examined.

Group H

The patient who harbored Group H streptococci showed the organisms in only one of three cultures. This patient had exudative tonsillitis and pharyngitis. There was little injection around the areas of exudate, however, and the cervical lymph nodes were not enlarged or tender. The white-cell count was 5500.

Group L

The patient who harbored Group L streptococci in the throat and developed antibodies during convalescence entered the hospital because of feverishness, cough, hoarseness and nasal discharge. He appeared only mildly ill, and the oral temperature was 101°F. The soft palate was slightly edematous but not injected. The tonsils had been removed. The pharynx showed only slight redness, and the cervical lymph nodes were neither enlarged nor tender. Fine rales were heard over the right lower posterior portion of the chest, and a roentgenogram showed a small area of pneumonia above the right diaphragm. The white-cell count was 15,800. The only throat culture obtained was at the time of admission; Group L streptococci were identified. After two days in the hospital the temperature became normal, and the pulmonary infiltration disappeared by the sixteenth day. The clinical diagnosis was primary atypical pneumonia.

The antistreptolysin titer measured 200 units on admission, 250 units on the fourteenth day and 317 units on the twenty-ninth day. The antifibrinolysin titer, which on admission was 100 units, was 200 units fourteen and twenty-nine days later. Since there is no information whether Group L streptococci produce streptolysin or fibrinolysin, and since adequate cultures were not obtained to rule out the presence of some other group, it was impossible to determine if this illness was due to Group-L streptococci.

Discussion

With the exception of scarlet fever and puerperal infection, information concerning the importance of the different Lancefield groups of beta-hemolytic streptococci in various types of infections is meager. Scarlet fever is rarely caused by streptococci of groups other than Group A. Hare,[11] in a summary of several investigations, reported that 115 patients with scarlet fever showed Group A streptococci and that in 2 cases the grouping was unknown. Gardner[12] isolated a Group G and a Group F streptococcus respectively from 2 of 21 patients with the disease.

Apparently beta-hemolytic streptococci of groups other than Group A are frequent causative agents in puerperal fever and septic abortions. In Hare's[11] summary of 206 patients with these infections, 3 cases were caused by Group B, 3 by Group C and 1 each by groups D and G and ungrouped organisms, the remainder being associated with Group A streptococci. Hill and Butler[13] reported 110 cases of beta-hemolytic streptococcus infection of the uterus, 82 of which were caused by Group A, 12 by Group B, 10 by Group C and 6 by Group G. Thus, approximately 25 per cent of these patients had infections caused by beta-hemolytic streptococci not belonging to Group A.

In erysipelas there is some indication that Group C plays a significant role, since in 28 cases studied, this organism was isolated in 5 cases.[11]

Further data indicating the importance of streptococci of groups other than Group A are obtainable only from isolated case reports and cultural surveys of hospitalized patients. The report of one or several cases — or even of an occasional epidemic — due to the rarer groups of beta-hemolytic streptococci is of little aid in the estimation of the frequency with which the various groups cause disease. Likewise, reliable data are difficult to obtain from reports on cultures obtained from hospitalized patients with various types of infections, since the isolation of one of the groups of streptococci, especially from the oropharynx, does not necessarily indicate a causal relation to the pathologic process.

In the present study several sources of information were used to determine the role of the groups of beta-hemolytic streptococci in respiratory infections. These included not only cultures of all patients with respiratory infections admitted to the hospital from

certain selected organizations of recruits but also surveys among the healthy soldiers in the same organizations. Thus, the differences in the prevalence of beta-hemolytic streptococci among the healthy soldiers and those hospitalized with respiratory disease should indicate the group or groups responsible for the illnesses. The carrier rate for all beta-hemolytic streptococci in the healthy soldiers was about 9 per cent, whereas 14 per cent of the hospitalized soldiers carried these organisms. The difference in rates was due almost entirely to Group A streptococci.

By the additional use of serologic, clinical and epidemiologic data, segregation of the specific patients with respiratory illness due to the beta-hemolytic streptococcus was possible. That such a method was valid was shown in a previous study[4] and again by the data presented above. If all the hospitalized patients who developed either antistreptolysin or antifibrinolysin during convalescence are excluded from the cultural surveys, the carrier rates in the healthy and hospitalized soldiers were approximately the same. This indicates that the beta-hemolytic streptococci in the oropharynx of the majority of the patients with respiratory illness who did not develop antibodies were not causally related to the infectious process.

On the basis of the demonstration of antibody formation during the convalescent period, 38 per cent of all cases of respiratory disease that showed beta-hemolytic streptococci were cases of streptococcal infection. At this Army hospital, beta-hemolytic streptococci were recovered from 465 of 3026 patients with respiratory infections. Thus, serologically proved cases of streptococcal disease accounted for approximately 6 per cent of the respiratory admissions in recruits over a three-year period. It is to be emphasized, however, that during this period there were no large epidemics and that the majority of streptococcal illnesses occurred sporadically.

The serologic reactions observed in the serums collected from these patients harboring different Lancefield groups of beta-hemolytic streptococci were of considerable interest. Streptolysin O is known to be produced only by organisms belonging to groups A, C and G.[9] It is well established that Group A infections are followed by an increase in the antistreptolysin titer in the majority of cases. Antibody studies have rarely been employed in the analysis of infections caused by other groups. Kirby and Rantz[14] described an increase in antistreptolysin in the serum of a patient with bacteriemia due to a Group C streptococcus. In the present study 7 patients harboring this organism and 5 with Group G streptococci developed antistreptolysin during convalescence. Longcope[15] studied a thirteen-year-old boy with an infection due to the Group F streptococcus whose serum exhibited an increase in antistreptolysin titer. No case of a rise in these antibodies, however, was observed in the 11 patients harboring Group F strains reported above.

The antifibrinolysin response to infection by streptococci belonging to Group A have been described elsewhere.[16] In that study a correlation was demonstrated between the ability of the homologous strain to produce fibrinolysin in vitro and the occurrence of an antifibrinolysin response following infection. In the present study 1 of the 7 patients with infections due to Group C organisms and none with infections due to those of Group G exhibited a significant increase in the antifibrinolysin titer. This low incidence of antifibrinolytic response was expected, since both organisms usually produce less fibrinolysin than Group A.[10] The median in vitro production of 452 Group A strains was 117 units, of 137 Group C strains, 61 units, and of 104 Group G strains, 20 units of fibrinolysin.

In several of the patients with infections due to groups C and G the serums in the acute and convalescent phases were tested with fibrinolysin made from Group A, Group C and Group G streptococci. With the use of quantitative methods,[8] no significant differences in the titers of antifibrinolysin were demonstrated with these three preparations. These results, as well as those of a more detailed study,[17] show that fibrinolysins produced by beta-hemolytic streptococci of groups A, C and G are immunologically identical. Kirby and Rantz[14] found that the antifibrinolysin titer following an infection with Group C was approximately the same when measured by either Group A or Group C fibrinolysin. They suggested that, from a qualitative standpoint, these fibrinolysins are similar.

On the basis of serologic tests, 90 per cent of the streptococcal illnesses were due to Group A, 4 per cent to Group C and 3 per cent to Group G. A detailed study of the infections caused by streptococci of groups A, C and G failed to show any outstanding differences in the clinical pattern. The illnesses produced by each group were mild and of short duration. A few of the infections included as cases of infection with Group C or G may have been due to Group A, since in some the number of cultures obtained were not adequate to rule out the presence of the latter organism.

The role of beta-hemolytic streptococci of groups B, F, H and L in the respiratory illnesses could not definitely be ascertained. There was some clinical evidence that groups B and F occasionally produce an infection, since several patients exhibited such clinical features as a discrete exudate over the tonsils, diffuse redness and edema of the soft palate, tonsils and pharyngeal mucosa and enlargement and tenderness of the cervical lymph nodes.

Summary

A study undertaken to show the importance of the Lancefield groups of beta-hemolytic streptococci in mild respiratory infection is reported. In a large

Army camp, only 6 per cent of cases among patients admitted to the hospital from an organization of new recruits were due to streptococcal infections. Bacteriologic and immunologic studies demonstrated that 90 per cent of the infections were due to Group A, 4 per cent to Group C and 3 per cent to Group G. Clinical and epidemiologic evidence suggested that a few patients had infections due to groups B and F.

The illnesses produced by the various groups of beta-hemolytic streptococci were similar. Streptolysin O antibodies were demonstrated in serum specimens obtained from patients harboring organisms of groups A, C and G. Antifibrinolysin developed in patients with infections due to Group A and Group C streptococci. The titer of antifibrinolysin in the serum specimens taken during the acute and convalescent phases from patients with infections due to groups C and G was identical when measured with fibrinolysin prepared from organisms of groups A, C and G.

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TRIPLE HEMISECTION: A SIMPLIFIED PROCEDURE FOR LENGTHENING THE ACHILLES TENDON*

Lieutenant Colonel Rafe Nelson Hatt, M.C., A.U.S., and Major Timothy A. Lamphier, M.C., A.U.S.

About twenty years ago, a new procedure for lengthening the Achilles tendon was first employed. It is known as triple hemisection. Dr. Michael Hoke, who is believed to have been the first to use this simplified technic, did not publish his results. And yet, according to White,† "lengthening the heel cord is the commonest single operative procedure in the repertoire of most orthopedic surgeons, and the reporting of any simplification of the technique seems justified. . . ."

Our purpose in describing this operation is to reemphasize its simplicity. Excellent results have been obtained without the necessity of using the so-called "open tenoplastic technic." Certainly, the much shorter operative time alone indicates triple hemisection of the Achilles tendon as the procedure of choice. Surgical exposure of a large portion of a tendon or of any internal structure necessarily carries with it the possibility of postoperative sepsis. Even the availability of such new drugs as penicillin and streptomycin does not mean that infection of tendon sheaths is easy to control. Another and even more important consideration is that the continuity of the tendon is maintained.

A so-called "tenotome" (Fig. 1), is used in this procedure. Its outstanding feature is a narrow thin blade that requires only a minute puncture hole in the skin prior to piercing the substance of the tendon. Once the knife has been inserted into a tendinous structure, there is no danger that the blade will be separated from the handle. Since the blade

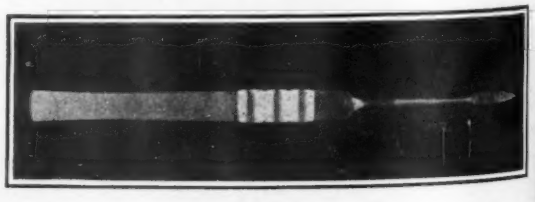

Figure 1. *Photograph of Tenotome.*

*From the Cushing General Hospital, Framingham, Massachusetts.

†White, J. W. Torsion of Achilles tendon: its surgical significance. *Arch. Surg.* **46**:784-787, 1943.

is extremely narrow, it is easy to maneuver, and there is less danger of destroying surrounding structures.

The usual type of Achilles shortening found in Army patients may be attributed in the main to casts applied to the lower extremity with the foot in some degree of plantar flexion. Also, following compound comminuted fractures, even though the foot has been encased in plaster in a neutral position, the tendon or belly of the gastrocnemius muscle becomes scarified and tends to contract, with subsequent shortening. Occasionally, after peroneal palsy, patients fail to wear dropped-foot or night splints, with the result that a shortened tendon often develops. These conditions are opposed to the usual civilian type of postpoliomyelitis in which muscle imbalance results in shortening of the Achilles tendon.

White has described one of the main problems in elongating the Achilles tendon as follows: ". . . the explanation of the difficulty encountered in lengthening the heel cord by simple subcutaneous tenotomies lies in the fact that there is approximately a ninety-degree twist of the structure on its own axis within the surgical field, and that when even two thirds of the medial fibers have been severed at one level and two thirds of the lateral fibers at another level, enough fibers miss being cut to cause the tendon to 'hang'." This statement describes the tendinous arrangement most adequately. This anatomical fact regarding the tendon of the gastrocnemius muscle is not, however, well known and bears repetition.

The operative procedure is illustrated in Figure 2. The lower extremity is properly draped. A blood-pressure cuff may be applied about the midthigh level and inflated to a pressure equivalent to 600 mm. of mercury; but its use is optional. The plantar surface of the foot is then placed against the abdomen of the operator, so that the Achilles tendon is stretched. Thus, both hands of the surgeon are free to carry out the procedure, and at the same time, tension of the tendon is maintained. With the use of the fingers of one hand to estimate the approximate width of the tendon, a stab wound on the medial aspect of the extremity is made into the substance of the tendon about 2.5 cm. superior to its insertion into the calcaneal tuberosity, with the flat of the knife blade on the same plane as the longitudinal fibers of the tendon. Greater success is assured if, when the tip of the blade has pierced the tendon, its course is altered from a simple transverse to a posterolateral direction. Because of the twist in the Achilles tendon, as demonstrated by White, at least a third of the tendon at this level must be divided. Caution must be taken to follow the medial approach, which alone gives assurance that the posterior tibial nerve or vessels will not be injured. This point cannot be too strongly emphasized.

Once the operator is certain that the point of the blade has passed through to the lateral aspect of the tendon, the knife is turned through an arc of 90° in an anterior direction. Provided that this maneuver

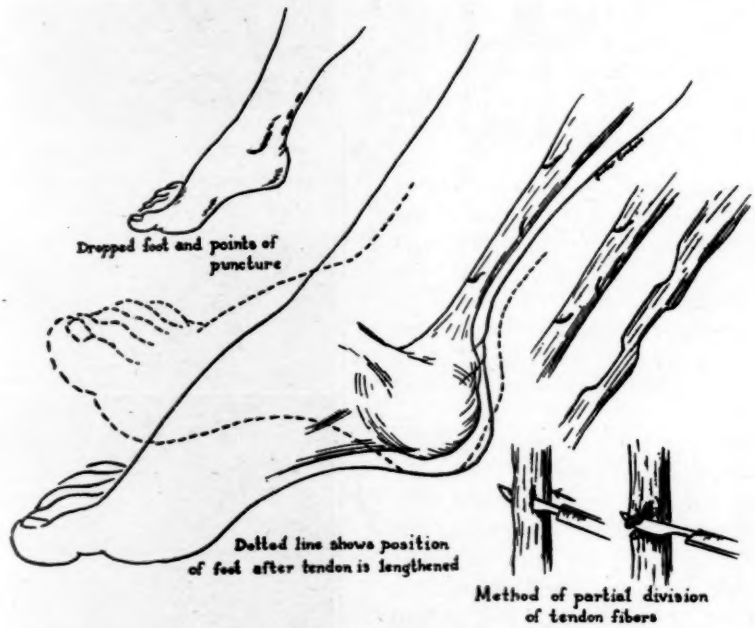
Dropped foot and points of puncture
Dotted line shows position of foot after tendon is lengthened
Method of partial division of tendon fibers

FIGURE 2. *Diagrammatic Representation of the Operative Procedure in Triple Hemisection of the Achilles Tendon.*

has been carried out properly, approximately one third of the tendon will have been divided.

When a second operative site, about 2.5 cm. above the original stab wound, has been selected, the tenotome blade is again passed by a medial approach through the skin and into the substance of the tendon. The course of the blade is again altered from a simple transverse to a slightly anterolateral direction. After the operator has determined that the tip of the blade is felt subcutaneously on the lateral aspect of the extremity, the blade is rotated through an angle of 90° posteriorly, and again approximately one third of the structure will have been divided.

The third — or most proximal — puncture wound is made about 2.5 cm. above that just described, the tenotome blade passing through the substance of the tendon in a posterotransverse direction. Again, the blade is turned through an angle of 90° anteriorly, and the operator is conscious that the fibers have been divided.

The next maneuver consists in the exertion of greater pressure against the plantar surface of the foot opposite the metatarsal heads. The weakened tendon, which relaxes as the fibers slide apart, can be lengthened until the foot is dorsiflexed to an angle greater than 90°. One should not be too enthusiastic in obtaining a markedly acute angle in dorsiflexing the foot, since the tendon may be completely divided and its continuity lost.

The entire procedure can be completed within a few minutes by an experienced operator. The assurance that the continuity of the tendon has been maintained is of primary concern. The results obtained by this simple method leave open to doubt the desirability of other, more prolonged procedures.

After the dorsiflexion of the foot to the desired angle, the three puncture wounds are dressed with sterile gauze. Sutures are entirely unnecessary, for the wounds are so small that the skin edges do not require approximation. With the foot maintained in the dorsiflexed position at all times and with the leg fully extended to 180° at the knee joint, the extremity is encased in plaster from the toes to the lower thigh level. Jack-knifing of the extremity at the knee joint while the cast is being applied must be avoided, to maintain tension on the Achilles.

An inexperienced operator may completely bisect the tendon, and when this occurs, a modified open tenoplastic repair must be resorted to.

Postoperatively, the cast is not removed in less than four weeks, to prevent recurrent shortening of the tendon. From the fifth through the eighth week the patient is allowed to remain ambulatory during the day, but a posterior plaster shell or a so-called "night ladder splint" should be worn while the patient is sleeping.

The double-exposure photographs (Fig. 3) represent the degrees of dorsiflexion before and four weeks after operation. Shortening of the Achilles tendon in this case resulted because the patient had previously had a cross-leg skin flap applied to his heel, and the foot had been left in a flexed plantar position for several weeks.

* * *

The triple hemisection method of Dr. Michael Hoke for lengthening the Achilles tendon has been described in detail. Difficulties arise in attempting the procedure in cases in which unsuccessful subcutaneous tenotomy has been attempted at some previous date. Occasionally, inexperienced operators attempt to gain too great an angle of dorsiflexion and completely disrupt the tendon. It should be remembered that, in marked deformities, the puncture wounds of the heel cord should be proportionately separated farther apart to obtain greater

FIGURE 3. *Double-Exposure Photographs Showing the Degree of Dorsiflexion before (upper) and after (lower) Operation.*

lengthening and thus to correct the greater degree of plantar flexion.

One criticism of this technic is that the ruptured fibers tend to become adherent to surrounding structures. This has not happened, however, in the hundreds of lengthening procedures carried out as described above. Certainly, the open tenoplastic procedure traumatizes and exposes a greater amount of subcutaneous structures than the simpler triple hemisection. One of us (R. N. H.) has had the opportunity to use this technic frequently during the past twenty years. The results obtained have been excellent and confirm the value of this simple method.

PRURITUS ANI*

NEIL W. SWINTON, M.D.†

BOSTON

A REVIEW of the voluminous literature on the subject of anal pruritus reveals that nearly as many theories regarding the etiology of this condition have been advanced and methods of treatment advocated as there have been authors. Several conclusions might be taken from this. First, the final answer has not been written, and there may be several etiologic factors involved in many of the patients with this condition. Secondly, because so many preliminary good results have been reported following so many apparently different types of therapy directed primarily toward the perianal region, the treatment of the patient, in addition to the management of the local anorectal condition, may be an important factor. Thirdly, there may be a factor common to many of these apparently different types of treatment that has been overlooked by the attention given the variety of drugs, injections and surgical procedures used on the perianal area.

The fact that many of these methods are not permanently successful is apparent in that a large number of the patients seen at the Lahey Clinic have already had some type of local anorectal therapy for anal pruritus without more than temporary relief.

It is not the purpose of this paper to announce that a cure for every patient with anal pruritus has been discovered or to present a long statistical study, but it is a condition that is often seen and one in which we have been particularly interested for several years. Many of the studies reported in the literature have not been carried out, and there are many types of treatment that we have not utilized; it is a frequent complaint, however, and many of the intractable cases present serious and complicated problems.

As a result of this experience we have developed certain theories regarding the etiology of this condition and methods of treatment. It is the purpose of this presentation to discuss the factors that may give rise to anal itching or may cause an anal pruritus to become an intractable and definite pathologic entity, and to present the methods of treatment that have proved most satisfactory.

ETIOLOGY

Some precipitating factor begins an anal pruritus. An anal itching is often noticed following a period of diarrhea or constipation, particularly if there is some associated anorectal disease. It is also a frequent observation that fatigue, worry and nervous tension are associated. Usually, this itching is transient and causes only mild and temporary discomfort.

The natural tendency for relief of an anal itch is to rub or scratch, which, if continued, causes local congestion, trauma and irritation and allows the introduction of infection into the perianal skin and adjacent tissues. This, in turn, causes more itching and sufficient excuse for further scratching and irritation of the part. Thus, a cycle of scratching and itching is established that, in the long-standing, intractable case, may develop into such a habit fixation that the patient is powerless to let himself alone and is awakened from a sound sleep, scratching himself.

This scratch-itch syndrome is a basic factor in the background of a large number of patients with an intractable anal pruritus and must be thoroughly appreciated by both the physician and the patient. Patients with an intractable condition of this type who have been treated by several physicians with various remedies often state that no one had ever taken the time to emphasize the importance of the avoidance of irritation to the parts affected. It is my belief that many of the intractable cases would never have developed if this single principle had been followed in the early stages of the disease.

Scratching is not the only cause of pruritus ani — it is an important factor, particularly in intractable cases, but it must not be overlooked that something additional caused the original itch. In

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From the Department of Surgery, Lahey Clinic.

†Surgeon, New England Baptist, New England Deaconess and Massachusetts Women's hospitals; surgeon, Lahey Clinic.

intractable cases this precipitating factor may have disappeared or become so altered by the passage of time and the development of local perianal changes that it is no longer significant, but it must be remembered that at one time there were factors favorable for the development of an itch in this region. It must also be recognized that the incidence of recurrence in this disease is high following alcohol injections, undercutting operations and other procedures in which a definite cessation of itching and relief from the tendency to scratch have been established, indicating that there are other underlying factors than the mere scratch-itch syndrome.

A wide range of factors may be responsible for the development of anal pruritus, and in long-standing intractable cases many factors may be involved in a single patient. Stokes[1] has divided into five groups the factors that should be considered in establishing the etiology of a given case of anal pruritus. These are considered below.

Physiologic Factors

The anorectal region contains a sweat apparatus called the apocrine system, which, similar to the axillary, submammary and periumbilical areas, is a part of the sexual glandular system and has, among others, two important characteristics: the sweat contains protein and excess carbohydrate and is more alkaline than ordinary sweat, and the apocrine sweat glands are peculiarly responsive to emotion, particularly sexual tension. This area, with its high-protein and high-carbohydrate content and alkalinity, is thus an ideal medium for the introduction of fungous infections and secondary invaders.

The physiologic state of the gastrointestinal tract itself may be important. It is not unusual to find that a stubborn constipation or diarrhea — owing to an atonic or spastic bowel — aggravates an anal pruritus.

There has been considerable discussion on the effect of intestinal flora on this condition. We have had little experience with studies of the intestinal flora and, in general, have not been impressed with the importance of this factor. We have had no experience with the use of autogenous vaccines. It has seemed sufficient to establish a normal bowel function, and adequate treatment of an irritable colon has been of value in the treatment of many stubborn cases.

It has been noted that many soaps are irritating in this condition. In patients who have a deficient amount of fat in their normal skin secretion, the excessive use of soap often causes local irritation. The sodium salts of saturated fatty acids are relatively irritating, and the sulfite in certain toilet tissues has been reported as a frequent source of irritation.

Excessive carbohydrate intake not only predisposes to an increased alkalinity of the part affected but also causes an increased hydration of the skin. Excessive fruit juices may have the same effect. Alcohol, as a peripheral dilator, may be significant.

Infective Factors

The role of fungous infections in anal pruritus has received considerable attention.[2] Terrell and Shaw[3] reported an incidence as high as 60 per cent in cultures taken from the perianal area. In the few patients in our series on whom careful bacteriologic studies were made, the incidence of fungous infections was not high. Pyogenic flora, with occasional hemolytic streptococci, were the usual findings. Stokes[1] has pointed out that a variety of organisms may be associated with the disease, many of which, generally considered nonpathogenic, seem to become pathogenic for the patient concerned.

Allergic Factors

An allergic background has been encountered in a few of our patients with anal pruritus. Frequently, however, the exact relation of the anal itching is difficult to evaluate. Certain patients exhibit an apparent susceptibility to certain food allergens. A contact sensitivity to mercury, found in many of the surgical lubricants, and the absorption of phenolphthalein, frequently used in laxatives, may cause local sensitization and pruritus.

Psychogenic Factors

The importance of psychogenic mechanisms should never be overlooked in anal pruritus. Nearly every patient with an intractable anal itch presents a history of episodes causing tension. The relation of fatigue and worry can be noted in every patient and financial upsets, family maladjustments and frustrations are frequently observed. A patient with a long-standing anal pruritus usually develops a fixation on the anal area to such an extent that any upsetting influence to his existence is reflected by an anal itch, and his entire attention may be directed either to the actual itching or to the anticipation of the next episode of difficulty. This fixation may become so serious that the patient is on the verge of complete mental collapse. Such patients are desperate for lack of rest, are completely incapacitated and require immediate and serious attention.

Stokes mentions the anal erotic type of anal pruritus, which we have encountered in a small number of patients, usually young men. Sexual problems may enter into many of these cases.

Anal pruritus was an extremely rare disease in the Armed Forces as compared with its incidence among civilians. In my experience, which was largely confined to combat infantrymen but included many thousands of hospital admissions, I recall only one patient presenting an intractable anal pruritus; he was obviously maladjusted, and his one desire was to be transferred out of the

combat zone. The majority of these men, experienced infantrymen, had the highest morale and were too busy and occupied most of the time to develop an anal fixation and spend their time worrying and itching and scratching. It must be remembered that these men were frequently without baths or clean clothes for days at a time and were in a hot, humid climate.

Mechanical Factors

Nearly every case of anal pruritus is precipitated by some local mechanical factor. If this itching begins when certain other favorable factors exist and relief is not obtained, the condition may become intractable.

Minor trauma to this area is not unusual. The act of defecation may cause an abrasion or a fissure. The effects of instrumentation, rubbing or scratching may begin an itch. Hemorrhoids may be responsible, but probably less frequently than has been believed. Large skin tabs, however, by rubbing together, causing congestion and interfering with mechanical cleanliness of the area, often seem to be important.

A contracted anal ring, a condition referred to in the literature as pectenosis, is a frequent factor. Infected or inflamed crypts are rarely involved, in our experience.

Treatment

A general physical examination, including an anoscopic and proctoscopic examination of the lower bowel, should be done in all cases. Routine blood counts and a urine examination should be carried out. In the majority of cases, after examination, the cause will be obvious, and the treatment apparent. In many, however, further laboratory and roentgenologic studies will be required and possibly the help of the dermatologist, internist, gastroenterologist or psychiatrist. It is to be noted that the original or precipitating factor in the long-standing case of anal pruritus may have disappeared and, from the standpoint of therapy, may not be particularly significant.

For constitutional treatment, the most important requirements are adequate rest, relaxation and relief from tension. Hospitalization and temporary but adequate sedation may be necessary in severe cases. A small amount of a combination of a tincture of belladonna and luminal, particularly when there is an associated spastic colon, is a helpful sedative. Chloral hydrate ensures a good night's sleep. A normal bowel function should be established as early as possible, and the intake of carbohydrate, excess roughage and alcohol should probably be reduced.

Excessive sweating, which may be troublesome, is the only indication for radiation therapy, since in our experience cases in which such therapy was used routinely nearly all recurred after a period of six to eight months of temporary relief, and it was found that they were invariably much more difficult to control. We have observed cases in which cancer developed in the anal area from the excessive use of radiation therapy. At the present time it is rarely used.

The presence of seborrhea, psoriasis, eczema, contact dermatitis and certain other strictly dermatologic conditions should not be overlooked. Special studies, particularly for food allergies, may be necessary in certain stubborn cases.

In considering local therapy, we are in entire agreement with Stokes that too much attention has been given to this phase of the management of the patient with anal pruritus. Most patients with long-standing intractable anal pruritus have had one or more series of radiation treatments, undercutting operations or alcohol injections, hemorrhoidectomy, cryptectomy, rectal dilatation or some other surgical procedure. They present a scarred anal ring and perianal area that has obviously been made worse by such treatment. Many of these patients had never been instructed to keep themselves clean and stop scratching.

Several years ago, while doing an alcohol injection on a patient with anal pruritus, I found a visitor very much interested in the procedure and in the disease. She was a missionary who had been in charge of a hospital in India for many years and had never seen a native with an anal pruritus of the type described in the literature, the explanation, in her opinion, being the fact that the Indians had never known modern toilet tissue and merely washed themselves frequently. Certainly, adequate local hygiene is the basis of any local therapy for anal pruritus. That this is not the only factor, however, was borne out by my experience in the Army referred to above.

Our patients are routinely advised to wash themselves often with cotton moistened in warm water and to eliminate toilet tissue and soap. They are instructed never to touch themselves with their fingers or any object other than cotton. The ensuing dryness and cleanliness and the avoidance of trauma will indefinitely control many of the mild cases of anal pruritus.

Among the local medications, potassium permanganate, in a strength of 65 mg. to 1 liter of hot water, is used almost routinely. Patients with a mild pruritus are instructed to sit in a basin of this solution for fifteen to twenty minutes at a time as often as is necessary to control the itching. The usual cause of failure in the use of potassium permanganate has been that the solution has not been employed for a sufficient period. The application of a 1 per cent solution of phenol in calamine lotion may be all that is necessary to relieve the difficulty. In the severer cases, continuous wet dressings and hospitalization may be required. Burow's solution, containing 1 per cent alum and

3 per cent lead acetate, is mild and helpful. Castellani carbolfuchsin paint — containing carbolfuchsin, phenol, boric acid and water, with or without resorcinol — is satisfactory in many cases; at first it should be diluted 1:2 and 1:3. Extremes of heat or cold may give prompt relief in certain acute situations.

In general, nonirritating, bland solutions are used and greases and ointments avoided. When lubrication is required, castor oil or olive oil may be helpful but should be employed sparingly. Frequently, following the continued use of potassium permanganate soaks, the skin tends to become dried out and mildly irritated. If actual fissures develop, ammoniated mercury ointment or a 2 per cent silver nitrate solution may aid healing. The coal tar preparations are at times indicated.

There are definite indications for surgery in these patients, but as our experience with this condition has increased, surgery has been used less and less. Surgery should be directed toward the removal of sources of infection and the correction of obvious local disease. Infected crypts should be drained, a contracted anal canal should be corrected and large edematous skin tabs that interfere with local hygiene of the area should be removed. The routine removal of hemorrhoids relieves few patients. Anal fissures should be excised, and fistulas should be removed.

At a certain period in our experience with this condition, a number of the more serious cases were treated with alcohol injections. Both the technics described by Stone[4] and Buie[5] were used. Temporary relief was obtained in all patients. Permanent relief was apparently obtained in a high percentage treated with the Buie technic, particularly when sufficient alcohol had been given to cause an actual slough, with complete destruction of the skin. The excessive scarring resulting from many of these procedures, however, was certainly not desirable. We have had a limited experience with the injection of local anesthetic-in-oil preparations. Temporary relief of anal pruritus has also been obtained following the injection of large quantities of both saline solution and distilled water. We have had no experience with the local injection of oxygen, with any of the tattooing procedures or with the Ball undercutting operation or any of its various modifications.

It has seemed to us that one factor common to all these different procedures has not been sufficiently emphasized, namely, local anesthesia to the affected part. This gives definite relief from the scratch-itch syndrome and enables the patient to rest. The only real justification in these radical procedures is this common denominator. They should be reserved for the severe cases in which rapid complete rest and relaxation are imperative. It is our firm belief, however, that the incidence of recurrence will remain high if complete reliance is placed on these surgical procedures, and adequate attention is not given to the various factors that may be in the background and to the restoration of the perianal skin to its normal tone and consistence.

* * *

When patients with anal pruritus are first seen they should be given a brief but intelligent discussion of the subject. In this way, the patients themselves are often able to suggest etiologic factors that obviate expensive study and consultation. The patients must understand that they are faced with what may be a chronic condition. They must learn to treat themselves and to forestall recurrence of the anal itching. It should be pointed out to them that rest, relaxation and freedom from nervous tension are essential for permanent relief and that the object of local treatment is to eliminate infection and to restore an irritated, infected area to its normal state. When surgery is indicated, the definite limitations of the surgical procedure and its object should be pointed out. In many patients who present themselves with a long-standing anal pruritus for which they have been promised a cure following surgery, no attention has been given to any of the other factors that may have been responsible for the disease. The physician who regards the patient with an intractable anal pruritus as a neurotic and gives him no attention deserves little sympathy; the latter has a real disease that can be serious and disabling, but with a reasonable appreciation of the responsible factors and an intelligent approach to the treatment of the patient and the local condition, satisfactory results for this distressing condition can be achieved.

605 Commonwealth Avenue

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MEDICAL PROGRESS

ABDOMINAL SURGERY (Concluded)

ARTHUR W. ALLEN, M.D.*

BOSTON

APPENDICITIS

Keyes and Cook[47] discuss the differential diagnosis in acute appendicitis in the presence of diarrhea. This combination occurs rarely but often enough to be kept in mind, especially in children or when the inflamed appendix is adjacent to the rectal wall. In appendicitis the pain is not relieved by the passage of a stool or flatus with or without enemas, whereas temporary complete relief is always thus obtained in gastroenteritis. Vomiting is more frequent and more prolonged in gastroenteritis, and the white-cell count is usually higher in appendicitis. Tenderness in the pelvis on rectal examination is diagnostic of appendicitis.

Fifty patients with proved appendiceal peritonitis were treated with massive doses of penicillin by Crile.[48] Half the cases were considered to be generalized peritonitis, and the others were localized. Eight patients had palpable masses at the time of entry, and 25 had masses during the course of observation. Twenty-three were treated conservatively; 22 had immediate appendectomy, and 5 had exploration only. After experience with smaller doses, Crile concluded that it was necessary to give 100,000 units of penicillin every two hours for two to six days, or until clinical signs had abated, followed by smaller doses for three or four additional days. The average period for the large doses was four days. Crile believed that the patients treated conservatively did better than those operated on immediately. A strict Ochsner regimen was carried out, with continuous gastric suction until normal peristalsis returned. There was a notable lack of ileus in the group. Masses disappeared under the treatment, and none had to be surgically drained; some of the pelvic abscesses, however, may have drained spontaneously through the rectum. Thirty-six per cent of the appendectomy wounds became infected, but were not tender and usually were not serious. There was one death in the group from mesenteric thrombosis. Experimental data are offered in support of the rationale for the large dosage used. This was based on the apparent inhibiting influence of *Escherichia coli* on the effect of penicillin.

It is quite probable that future reports on the use of streptomycin in such cases may be even more spectacular. It should be borne in mind that Crile's patients had accepted methods of treatment hitherto found useful, in addition to the penicillin. Furthermore, the results in these cases, which occurred in military personnel, are compatible with earlier statistics concerning optimum recoveries for a similar group.

The differential diagnosis of acute appendicitis and acute, nonspecific mesenteric adenitis is ably presented by Aird.[49] Tabes mesenterica is more frequent in children and adolescents but may occur at any age. A history of acute infection — particularly of the throat — preceding the attack is often obtained. The pain is colicky, with relief between spasms. The temperature is usually higher, and there is more associated nausea and vomiting than is generally seen in appendicitis. The localization of the tenderness is above McBurney's point and slightly to the right of the umbilicus, discomfort on palpation extending from this region diagonally toward the left upper quadrant. This distribution of tenderness follows the course of the small-bowel mesentery. Discomfort on rectal examination is absent. The white-cell count is frequently lower than that usually found in appendicitis but may be high, with an increase in polymorphonuclear leukocytes. Aird suggests a virus infection as the etiologic agent, based on negative cultures from biopsied lymph nodes in the acute phase of the disease. He states that spontaneous recovery within forty-eight hours is the rule; a history of repeated attacks is frequent.

Although appendectomy does not appear to affect an acute mesenteric adenitis, it must be borne in mind that the differential diagnosis is not always possible. It is safer to explore many of these patients and remove the appendix than it is to overlook a true appendicitis with bizarre symptomatology and physical findings. Future attacks, if they occur, do not cause the same alarm to the patient, his family or the physician after the appendix has been removed. Furthermore, there is considerable evidence of a reduction in acute abdominal episodes due to mesenteric adenitis after appendectomy.

According to Etherington-Wilson,[50] 80 per cent of the cases of torsion of the great omentum are diagnosed preoperatively as acute appendicitis. The literature yielded 190 cases of this condition, and the author reports 4 cases of his own. Twisting of the

*Lecturer in surgery, Harvard Medical School; chief, East Surgical Service, Massachusetts General Hospital.

great omentum may be complete or partial, the latter at times being represented by a single strand. The strangulation of the blood supply may also be partial or complete. The torsion may be unipolar, representing the primary or idiopathic type, or bipolar from a secondary fixed point. There were 73 cases of the primary type, which were exclusively intra-abdominal and without known cause. The secondary rotations may be unipolar or bipolar, occurring as a result of omentum fixed within a hernial sac, adhesions or deformity and caused by past or present inflammation. The condition is usually acute but may be subacute or chronic. Men between twenty and fifty years of age are generally affected. A combination of factors is probably responsible. These include abnormal peristalsis, contractions of the abdominal wall and diaphragm, sudden twisting or jerky motions of the body, direct blows on the abdomen, omental disfigurations by tumors, overloading or uneven fat deposits, pedicle formation, scarring, raggedness, adhesions, bipolar attachments and displacement of the omentum during operations for abdominal tumors.

The Stomach and Duodenum

Ladd et al.[51] report the experience with congenital hypertrophic pyloric stenosis at the Children's Hospital, Boston. From 1915 to 1945 a total of 1145 cases were treated; 588 patients were admitted in the first twenty years covered in this report. There were 35 deaths in this group — a mortality of 5.95 per cent. In the ten-year period from 1935 to 1945, there were 577 cases with 5 deaths — a mortality of 0.89 per cent. There were no deaths in the last 225 cases. The value of preoperative preparation, ether anesthesia and the Robertson gridiron incision is emphasized.

Hobbs and Cohen[52] collected from the literature 40 cases of gastroduodenal invagination and presented an interesting case report of a sixty-one-year-old woman whose intussusception was due to a submucous lipoma of the stomach. Reduction and sleeve resection resulted in recovery. The authors stated that lipomas constitute about 3 or 4 per cent of all benign tumors of the stomach, and they found only one other report of invagination due to lipoma. Prolapsing mucosa, pedunculated adenomatous polyps, pancreatic rests and leiomyomas appear to be the most frequent etiologic agents.

Moses[53] collected 150 cases of diverticum of the stomach. The symptoms are similar to those of ulcer, and the diagnosis is made by roentgenographic studies. Relief is obtained by excision of the diverticulum. Walters[54] discusses the subject and adds 3 new case reports of this lesion. He refers to an earlier report of 2 cases from the Mayo Clinic.[55] Brown and Priestley[56] cite a case of diverticulum of the stomach associated with acute massive hemorrhage.

The experience with carcinoma of the stomach at the New Haven Hospital is reported by Lawrence and Kay.[57] They compare two series of cases in the decades from 1920 to 1940 and find that the patients did not report to the hospital earlier in the latter group; however, the resectability had increased, and the operative mortality had decreased. There was a 40 per cent loss owing to obvious inoperability or to the patient's refusal of operation. The lesions in 35.5 per cent of patients explored were not resectable; 8.8 per cent of patients were lost through operative mortality, whereas 9.3 per cent gradually declined and died after leaving the hospital. Of 208 patients admitted, 4.4 per cent were alive and symptom free at the end of five years, and 2 per cent died of other causes. There was a corrected salvage of 21 per cent in the 48 patients undergoing resection.

Reiteration of the importance to the physician and the layman of early diagnosis and radical treatment in cancer of the stomach is warranted. Temporary relief of indigestion by an ulcer regimen and advertised remedies is misleading. Every effort should be made to study these patients properly and to institute correct therapy before the tumor is out of bounds. The most frequent fault is the conservative treatment of middle-aged people for an ulcerative lesion of short duration. The relief of symptoms often seen without disappearance of the ulcer results in the false impression that benign ulcer is the cause, and the chance for cure is lost.

Waugh and Fahlund[58] report 77 cases of total gastrectomy by the abdominal approach from the Mayo Clinic, with an over-all mortality of 44.2 per cent. The mortality rate dropped to 3.2 per cent in 44 patients operated on between 1940 and 1943 but rose again to 20 per cent during 1943. The authors point out the value of experience in this field of surgery as being the chief factor in reducing the operative risk. Over 50 per cent of their patients who survived lived for more than two years.

Smithwick[59] reported 10 cases of total gastrectomy, with emphasis on an aseptic anastomosis between the esophagus and the jejunum by means of a special clamp he has devised. One patient was alive and well ten and a half, and another five and a half years after operation. There was no operative mortality in the 7 cases in which the aseptic technic was used.

Lahey,[60] in a discussion of Smithwick's paper, reviewed the experience with total gastrectomy by the abdominal approach in his clinic. In 92 cases 2 patients survived for over five years after this procedure for lymphosarcoma of the stomach. The mortality rate has been reduced materially in recent years.

Kelly[61] reported the case of a patient who was alive and well seven and a half years after total resection for carcinoma of the stomach. The patient developed primary anemia after some years and

was restored to health by the institution of liver and iron therapy.

Malignant lesions of the duodenum are discussed by Dixon et al.,[62] who found a total of 49 cases, exclusive of those arising in the papilla of Vater, in the records of the Mayo Clinic. Operation was performed in 45 cases, and 4 were revealed at autopsy. There were carcinomas in 44 cases, sarcomas in 2, a lymphangioendothelioma in 1 and associated carcinoma of the stomach in 2. Obstruction was the prominent feature in 38 patients, whereas anemia was the prevailing finding in 6. The mortality from exploration and palliative procedures was 22 per cent. Segmental resection was performed in 4 cases, and local excision in 1, with no deaths, and in the 4 patients subjected to radical resection, there was death in 1. One patient was alive and well six years after operation. Interestingly enough, another patient lived for seven years after gastroenterostomy before succumbing to the disease.

In a collective report on duodenal ulcer in England and Wales, Wood[63] states that there were 43,000 fatal cases of this lesion in the decade before World War II and that 200,000 new cases occur each year in those countries. He mentions the increase in ulcer during the London Blitz and emphasizes the mental and psychic elements in the etiology. He agrees that the problem is primarily medical, surgery being reserved for complications. Although extensive resection is regarded as the best method of treatment in properly selected cases, he has observed reasonably favorable results in a limited number of cases treated by the method of Somervell.

Somervell[64] describes his technic of interrupting five sixths of the blood supply to the stomach, in addition to posterior gastroenterostomy, as a method of treating duodenal ulcer. He compares 400 patients so treated with 300 who had been subjected to subtotal gastrectomy. He states that gastric acidity remained lowered for five years by his procedure, with only 1 case of anastomotic ulcer. There were 7 cases of stomal ulcer in the gastrectomized patients. This is similar to the early attempts to cure hyperthyroidism by progressive interruption of the blood supply to the gland. It must be borne in mind that, in interruption of the blood vessels to the stomach, the influence of the altered nerve supply may also have a bearing on the results.

Dragstedt[65] has revived interest in vagus resection for the cure of duodenal ulcer. Much enthusiasm is being demonstrated in this approach. The immediate results at the Massachusetts General Hospital in nonobstructing duodenal ulcer are impressive. If this operation, performed transthoracically, proves permanent, it will offer a simple solution to the problem. We are watching with considerable reservation the vagus interruptions below the diaphragm, because of the difficulty of exposure of the nerves and the limited segments that can be removed by this route.

Stomal obstruction following gastroenterostomy is discussed by Gray and Sharpe,[66] who stress the two types met — one developing in the first few days and mechanical in origin, and another occurring from the seventh to the tenth postoperative day. If the mechanical arrangement is at fault, it may need operative correction. In the later, functional type jejunostomy for feeding will take care of the situation. Experience and careful technic of operation are regarded as obviating most of these difficulties.

Alesen[67] has devised an absorbable fenestrated tube made of beeswax, petroleum jelly, powdered elm bark and merthiolate with a melting point of 85°C. to be incorporated within the gastroenterostomy stoma. He believes that early obstruction is the most important to overcome and that this framework is sufficient for this purpose. The tube, which disintegrates as the elm bark expands, disappears from the stoma in ninety-six hours.

Usually, early obstruction responds to decompression of the stomach segment by gastric suction through a nasal catheter. The late obstructions are extremely troublesome in my experience, occurring frequently in depleted patients. A proximal jejunostomy tube, whose tip lies in the stomach, has been used with complete satisfaction in over 300 cases. Minor complications have arisen, but none that offset the comfort and safety of the procedure.[68] No fatal cases have resulted from the use of this method of decompression. In many cases a jejunostomy for feeding is also used, particularly in patients with prolonged obstruction and resultant malnutrition.[69]

Three excellent articles on the feeding of patients in preparation for surgery and during convalescence have appeared. All present variations of mixtures containing skimmed milk, skimmed-milk powder, Amigen, eggs, soy-bean flour, sucrose and salt. Varco[70] believes that patients who have lost weight should be treated by the addition of 5000 to 6000 calories of this liquid nonresidue mixture daily to the hospital diet. He states that it requires ten to twelve days of preparation for a weight loss of 20 per cent, three weeks for one of 25 to 30 per cent and a month for a loss above 35 per cent. The liquid is kept beside the patient, who is encouraged to take as much as he will. If he cannot take it voluntarily, it is introduced through a nasal tube by gravity. After this preparation, patients appearing in a depleted condition withstood major operations on the stomach or colon much better than those in an earlier series not treated in this manner.

Riegel et al.[71] and Lund[72] have also outlined the nutritional requirements of patients, with particular emphasis on protein. The formulas vary slightly, and the daily caloric values are based on maintenance rather than on the correction of previous

weight loss, as stressed by Varco.[70] Many variations can be worked out, and some of the mixtures appear to be more palatable than others. The viscosity can be reduced to allow for jejunal feedings by the Murphy drip. Vitamins and liver extract can be added.

Fewer problems are involved in patients who can take nutritional mixtures or properly prepared food either voluntarily or by gastric or jejunostomy tube than in those who require intravenous feeding as the main source of nourishment. So far, there seems to be a limit to the amount of carbohydrate and Amigen that can satisfactorily be used by this route. Further purification of the constituents employed for intravenous feedings and a better understanding of their management are needed.

Custer and his associates[73] have studied the problem of the so-called "dumping stomach" following subtotal gastrectomy. They believe that the symptoms of weakness, generalized and unpleasant sensation of warmth throughout the body, a cold diaphoresis of the face (particularly of the forehead) and cardiac palpitation after eating are due to the rapid emptying of the stomach into the jejunum. They found that 5.6 per cent of 500 patients studied complained of one or more of these symptoms; some of them obtained relief by lying down after eating, and many were finally relieved. This ultimate relief is explained on the theory that the jejunum becomes adjusted to the early deposit of food, and it is recommended that patients be instructed to experiment with the following measures for relief: to lie down after eating, to take 0.024 gm. (3/8 gr.) of ephedrine with two or three soda crackers before meals, 30 cc. of cream or 45 cc. of olive oil before meals, six small meals daily, a dry diet, and 10 drops of dilute hydrochloric acid half an hour before eating.

All who have seen many patients after gastrectomy are at a loss to explain the variation in their state of well-being. We found that 87 per cent of patients with duodenal ulcer were well after subtotal gastrectomy.[74] Women were likelier to complain of weight loss and discomfort after eating than men. I am not convinced that rapid emptying of the stomach remnant is the whole explanation of these symptoms. To be sure, postprandial discomfort is a more frequent complaint after total gastrectomy than after subtotal, but many of these patients eat well and without distress and a few are hungry. There seems to be a reflex in some cases that may be based on changes in the autonomic nervous system. I am sure that many of the rapidly emptying stomach segments create no symptoms.

Perman,[75] of Stockholm, informs me that if these manifestations are severe and the patient is invalided by them, relief can be obtained by disconnection of the gastrojejunostomy and the establishment of a gastroduodenostomy. This appears to be a logical solution, since the food then passes over a more nearly normal route. The Billroth I procedure can rarely be done satisfactorily at the time of adequate subtotal gastrectomy for duodenal ulcer, but doubtless can be quite easily accomplished at a later date, when the elements of inflammation in the duodenal stump have disappeared.

The Small Intestine

A review of the literature by Williams and Walker[76] has revealed 300 cases of diverticulum of the jejunum. Two case reports of their own are added, one of which had been studied repeatedly over a number of years for pain and fullness in the upper abdomen, with negative findings. Finally, a diverticulum of the jejunum was diagnosed by roentgenograms, and relief of symptoms was obtained following excision of the lesion. The other patient was admitted with an acute abdominal condition and a pelvic mass. On exploration, bilateral ovarian cysts with twisted pedicles were removed, and a perforated diverticulum of the jejunum was excised. Other diverticulums were noted in a 50-cm. segment of the jejunum, which was successfully resected six weeks later. In both cases, on microscopic examination, the diverticulums contained all the normal structures present in the jejunum.

Atwood[77] observed 37 cases of Meckel's diverticulum in 69,000 admissions to the Truesdale Hospital. He presents an excellent review of the developmental background of the lesion and discusses the causes of small-bowel obstruction associated with it. He gives a case report of intussusception due to invagination of a Meckel's diverticulum.

Two cases of repeated massive hemorrhage, with the passage of bright-red blood by rectum, due to neurofibromas of the ileum, are reported by Baker and Halley.[78] After excision of these tumors, no intraluminal ulceration was revealed in the specimens. This feature is unusual, and it seems unlikely that such a large amount of blood could have been lost except through an open vessel within the lesion. Similar tumors frequently found within the stomach become umbilicated in the central portion, and these depressions lead to an easily recognized open artery if the specimen is removed during or shortly after excessive bleeding.

Moses[79] gives an excellent discussion on acute small-bowel obstruction. He stresses the fact that most of these patients are admitted to the hospital in an acutely depleted state and therefore should usually not be subjected to immediate surgery. Careful and intelligent attention to the correction of fluid and electrolyte balance and shock is the first consideration. In patients who cannot be brought out of shock, operation offers no additional hope for cure. Usually, these patients can be made ready for surgery in a period of hours, and preoperative preparation should not be prolonged beyond the optimum period for intervention. Moses reports 118 patients admitted, with 10 deaths; 90 were operated on, with

7 deaths. Of the 14 requiring resection of gangrenous segments, 3 died.

Foisie[80] emphasizes the high incidence of small-bowel obstruction occurring in veterans after recovery from abdominal wounds. This late complication is bound to occur frequently after the penetration and repair of the abdominal viscera.

Calihan, Kennedy and Blain[81] review the experience with acute intestinal obstruction at Johns Hopkins Hospital for a ten-year period. They found that 20 per cent of 204 patients succumbed. Fifty-five per cent of the deaths were due to peritonitis. The duration of the obstruction was a vital factor in recovery. Strangulation, followed by gangrene, and perforation were the primary causes of the fatal peritonitis. On the basis of experimental work in the same clinic by Blain and Kennedy,[82] Calihan et al. believe that antibacterial therapy, particularly with penicillin, will materially reduce the mortality in acute small-bowel obstruction. Dogs with isolated loops of small intestine treated with penicillin lived twice as long as control animals. With penicillin, the strangulated bowel became thickened, so that gangrene and perforation were delayed. They believe that their evidence points toward the bacterial theory, rather than toxic elements within the strangulated loop of bowel, as the chief cause of death.

Harris[83] reports the use of a single-lumen tube with a mercury-weighted balloon for decompression of the small bowel. This tube is of smaller outside dimensions than an effective double-lumen tube and yet has a larger lumen. He advocates the use of 4 cc. of mercury in the balloon and calls attention to the difficulty met in withdrawing this balloon through the pylorus. In two cases the mercury was spilled within the bowel without harm to the patient. The tube was occasionally allowed to pass intact through the rectum.

The members of the resident staff of the Massachusetts General Hospital have preferred this new tube to the Miller–Abbott tube. They find it easier to get through the pylorus and that decompression of the obstructed loops of bowel is more quickly accomplished. By using the laws of gravity, there has been little trouble in causing the mercury to flow in the right direction within the balloon when the tube needs to be removed. Less than 4 cc. of mercury is often enough, and this amount is seldom troublesome.

Another single-lumen weighted tube has been described by Cantor,[84] who recommends more and larger fenestrations in the tube. The high openings in the tube are designed to pick up secretions from the stomach, as well as at intervals along the intestine. It appears that the benefit of the principle of the decompression tube may be lost by this method, since the normal secretions above the obstructed loop contain valuable chemical elements that are absorbed if not removed. Also, this tube prevents the absorption of water, nutritious fluids and electrolytes that can often be administered while the decompression tube is in place.

The Colon

Schlotthauer[85] calls attention to the familial tendency in diverticulosis of the colon. He reports a family of 7 brothers, all of whom were afflicted with this ailment. Two sisters in the family did not have diverticulosis. Reports in the literature indicate the incidence of this disorder to be from 2 to 5 per cent of the population. Certainly, this figure is much too low for persons beyond middle age.

Meyer et al.[86] have given a good program for the preparation and aftercare of the patient in colon surgery. They cleanse the bowel with cathartics and enemas and then use succinylsulfathiazole (Sulfasuxidine) for several days. They take pains to bring about a chemical balance and adequate blood replacement. Spinal anesthesia is preferred. Open primary anastomosis after resection of the remaining colon was carried out in 25 patients, with 1 death from embolism. Early mobilization is stressed. Proximal decompression is omitted. Wounds are closed primarily with interrupted wire sutures. Patients averaged thirteen days in the hospital after operation.

Thomson and Daland[87] report the use of phthalylsulfathiazole (Sulfathalidine) in the preparation of patients with cancer of the colon and rectum, 1 gm. of the drug being given every four hours for five or six days preoperatively. The patients were usually well prepared for surgery, and the semisolid state of the fecal matter still in the colon was considered preferable to the more liquid effect of succinylsulfathiazole. In spite of the slipping back into the peritoneal cavity of the colostomy in 2 cases, one of which was not recovered for eight hours, there was only 1 death in the series of 49 cases. This fatal case occurred following resection of the splenic flexure after a cecostomy decompression for complete obstruction. The effect of the phthalylsulfathiazole was thereby thwarted, and death resulted from peritonitis. Sulfadiazine was given intravenously after operation. Primary anastomosis was done in all lesions of the colon.

Segmental resection in the left colon with primary aseptic anastomosis in 50 cases is reported by Waugh and Custer.[88] There were 2 deaths in the group. The Rankin three-bladed clamp was employed as the basis of the anastomosis. The hospital stay in this group was greatly reduced, the safety of the procedure demonstrated, and the short morbidity emphasized when compared with a large group of patients treated by exteriorization operations.

Another report of primary anastomosis following colon resection is presented by McMillan.[89] In 27 consecutive cases in which operation was performed, there was 1 death from pulmonary complications.

He stresses the use of two rows of interrupted nonabsorbable sutures in the anastomosis. The patients averaged eighteen days in the hospital.

It is interesting to those who have always used primary anastomosis following resections of the colon to find such a general interest in this procedure. Obstructive resections and other exteriorization methods have been satisfactory to many surgeons. It is creditable that the shorter period of disability from primary anastomosis has not increased the risk in the hands of surgeons using the various aids in preparation, anesthesia, suture material, operative technic and aftercare now at their disposal. I believe that delayed wound closure is a safeguard in addition to those mentioned above.

Olsson[90] emphasizes the need for careful examination of patients with cancer of the colon. The percentage of multiple lesions, in his opinion, is much higher than is usually believed. In one year, he found between 50 and 60 cases during examination of 700 or 800 patients. In 5 cases multiple lesions were found.

In 42 out of 220 patients with cancer of the colon and rectum, Sugarbaker[91] found involvement of other structures that could be removed. He reports a resectability rate of 78 per cent in a group of patients coming from a district where advanced disease, with weight loss and so forth, was the rule. Resectable extensions were found in the abdominal wall, uterus, urinary bladder, prostate, vagina, iliac vessels, ureter and femoral nerve. The vagina was involved more frequently than the prostate, probably owing to the thick fascial covering of the latter. Patients with other structures involved by continuity had symptoms of cancer of the bowel an average of fifteen months as against thirteen months in those without these extensions. The mortality rate in 72 patients in whom no other structures were involved was 9 per cent, whereas in the other group 19 per cent died; 19 patients with involvement to neighboring structures were alive two to five years after operation.

Young and Cole[92] present some interesting experimental evidence that intraperitoneal succinylsulfathiazole and phthalylsulfathiazole may be useful when the peritoneal cavity is contaminated by fecal matter. These drugs were absorbed more quickly and created less adhesions than other sulfonamides. No toxic reaction was evident in dogs receiving 1 gm. per kilogram of body weight. The proved effect on *Esch. coli* by the oral use of succinylsulfathiazole and phthalylsulfathiazole makes it logical to try these drugs intraperitoneally when indications arise. Young and Cole have treated a small group of patients with 0.1 gm. per kilogram of body weight without harm and suggest that larger intraperitoneal doses are probably safe.

It is interesting in this connection that Poth et al.[93] have found that penicillin inhibits the action of phthalylsulfathiazole and does not seem to affect the properties of succinylsulfathiazole, no explanation being available for this antagonism.

An excellent discussion on the management of the permanent colostomy is presented by Miles and his associates.[94] The daily colostomy enema or wash out seemed to be favored by the majority. A few preferred to let Nature take its course. Some believed that patients could train their colostomies to function at a certain hour each day. There was little support of the use of bags or cups. Miles seemed to give the most logical advice for these patients. He recommends a daily wash out of the colon with 750 cc. (1½ pints) of plain water at 80°F. He stresses the fact that hot water creates inertia of the muscular coat of the colon, with retention of part of the water within the bowel. Enemas should be introduced with the patient lying down, so that the muscles will be relaxed. He recommends finger dilation of the colostomy opening daily for a month, once a fortnight for two months and, finally, once a month for six months. After this, there is little tendency for contraction to take place. He uses a simple belt for support and disapproves of bags and cups, which produce prolapse, edema and bleeding of the mucous membrane.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 33051

PRESENTATION OF CASE

A sixty-six-year-old furniture crater entered the hospital because of exertional dyspnea.

Six years before entry, the patient had had an attack of pneumonia requiring four weeks' hospitalization, during which he received sulfadiazine, and rest at home for eight weeks. During this period he had gained 20 pounds in weight, which he had maintained. On resuming work he felt "better than ever before" but noted some exertional dyspnea and night sweats. He also had an occasional nonproductive cough and two or three paroxysms of coughing daily. Two months before entry, the patient saw a local physician because of continuing dyspnea and was referred to this hospital. During the summer before entry, he lost 8 pounds, which was not unusual for him during hot weather.

Physical examination showed a well preserved man in no discomfort. The only positive findings were scattered rales over the left lung base posteriorly.

The temperature was 99°F., the pulse 70, and the respirations 18. The blood pressure was 170 systolic, 90 diastolic.

Examination of the blood disclosed a hemoglobin of 14.0 gm. and a white-cell count of 15,500, with 83 per cent neutrophils, 5 per cent lymphocytes, 8 per cent monocytes and 3 per cent eosinophils. The nonprotein nitrogen was 25 mg. per 100 cc. The urine was normal.

An x-ray film of the chest showed the left hilus to be considerably increased in size and moderately elevated. Irregular linear bands of increased density extended outward into the upper middle and lower lung fields. The heart shadow was displaced to the left, and there was an inspiratory shift of the mediastinum to the left. The diaphragm moved fairly well. Within the density in the left hilus was a 2-cm. sharp line, suggesting a fluid level in a small cavity.

Bronchoscopy of the right bronchial tree was negative. The left main bronchus was normal, down to the upper-lobe orifice. Close to this orifice, which appeared slightly narrowed, there was narrowing of the left main and left lower-lobe bronchi, with some fixation and a slightly nodular appearance. Several biopsies from this area were reported as showing chronic inflammation.

On the eighth hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. EDWARD D. CHURCHILL: We are to discuss the case of a patient with a serious illness that led to operation. As the record stands, we must accept the facts that the symptomatology was of long duration and that all symptoms dated back to the episode of pneumonia six years before entry. It is always important to find out just what brings a patient to the doctor at the time that he summons the courage to make the trip. Nothing in the record, however, gives any lead to new symptoms or to an increase or change in old symptoms that might have led this patient to consult a physician two months prior to admission. The presenting symptom is recorded as "continued dyspnea." Even the significant fact of whether he had or had not raised blood is lacking.

DR. TRACY B. MALLORY: Can you answer that, Dr. Sweet?

DR. RICHARD H. SWEET: I am impressed by the difference between the story obtained in the hospital and that obtained in my office. I have the recollection that the patient had had recent symptoms of cough and occasional staining with blood, but I may be wrong.

DR. CHURCHILL: Is this fact, not being given in the history, admissible as evidence?

DR. MALLORY: Yes; I think you had better use all the information available.

DR. CHURCHILL: You will admit blood spitting?

DR. SWEET: My recollection is that the patient had had occasional recent spitting of blood, which was intermittent.

DR. CHURCHILL: Not every day, and occurring within the previous few months?

DR. SWEET: That was my impression.

DR. CHURCHILL: Consequently, there was a change that brought about a visit to a physician two months before admission. There is also no note of whether the sputum was examined in the hospital. We then focus our attention on a differential diagnosis between chronic infection and neoplasm of the lung.

May we see the x-ray films?

DR. JAMES R. LINGLEY: This is the enlarged left hilus that was described, with what looks like a fluid level in the central portion, outlining a cavity about 2 cm. in size. In addition, there are linear bands of density extending upward into the upper, middle and lower lung fields. The bands are also visible in this lateral view, extending backward to the base of the upper lobe and forward into the lower lobe. The heart is slightly displaced to the left side, and fluoroscopically a note was made that there was a mediastinal shift to that side on respiration.

DR. CHURCHILL: Where is the cavity on the lateral film?

DR. LINGLEY: The fluid level is not adequately demonstrated in the lateral view. I suspect that the cavity was posterior to the hilus, but I cannot be sure.

DR. CHURCHILL: Was the cavity peripheral?

DR. LINGLEY: I cannot be sure.

DR. CHURCHILL: Can you localize this process in the lateral film — whether it was in the upper or the lower lobe, or both?

DR. LINGLEY: I think that it involved both, but it was more marked in the base of the upper lobe.

DR. CHURCHILL: Do you think that this is a cavity far posteriorly?

DR. LINGLEY: That location in general.

DR. CHURCHILL: In this case the differential diagnosis lies, as I have said, between chronic infection with or without an acute exacerbation and a bronchiogenic carcinoma. I am not satisfied with the history as presented in the record, because there is nothing to suggest that a new entity, such as carcinoma, had come into the picture. If we admit the recent appearance of blood spitting, an increase in exertional dyspnea, some feeling of fatigue, some stiffness of the joints or a dozen other symptoms that might have taken this man to a physician two months before entry, I think that we are tipping the scale toward bronchiogenic neoplasm. I must raise one other question. In discussing another patient, Dr. Sweet recently stressed the point that an abscess cavity in the lung of purely inflammatory origin is usually peripheral, whereas in neoplasm it may be central. If we apply that yardstick to Dr. Lingley's interpretation that the cavity was centrally rather than peripherally placed, it favors carcinoma. This would have been a good case in which to try out examination of the sputum for tumor cells, but we are proceeding without sputum examination. The bronchoscopic findings were quite significant so far as narrowing and fixation of the bronchus were concerned. Such fixation is a sign that is associated with carcinoma rather than an inflammatory condition. There was no recent history of a bad cold or an attack of grippe, which might have exacerbated an old pneumonitis. A lung abscess appearing six years after pneumonia is unusual, and lacking further evidence I consider the scales weighed toward bronchiogenic carcinoma. I am willing to proceed with Dr. Sweet, if that was his diagnosis, with a thoracotomy incision for inspection and probable removal of the lung.

DR. MALLORY: Dr. Sweet, do you want to tell the operative findings?

DR. SWEET: Perhaps Dr. Benedict would like to describe the bronchoscopy.

DR. EDWARD B. BENEDICT: It is all given in the record. I did say "partial fixation," however—not complete.

DR. SWEET: I have been trying to obtain my office record because my impression was so different, and I regret that I did not review the history of the hospital record before it was presented. But, as I have said, my impression was that a definite change in the form of the cough or some expectoration with an occasional streaking of blood caused the patient to consult a physician.

I was not certain what the diagnosis was ahead of time. Dr. Benedict and I have often labored over cases of this type, and in quite an appreciable number we have been forced to perform an exploratory thoracotomy. As Dr. Churchill suggested, that is what we did. Before operation I was not certain whether the cavity was central or peripheral. My impression was that it was central, but it actually was peripheral. We made a thoracotomy incision and discovered that the most adherent portion of the lung was in the region of the dorsal division of the lower lobe; the adhesions were of the character that we have learned to associate with chronic infection. They were so dense that one could not develop a plane of cleavage. The lung had to be separated with a knife, but I did not believe that

there was a special indication for entering the extrapleural fascial plane. In separating that portion of the lung from the chest wall, however, I opened into the cavity, which presented the usual appearance of a chronic inflammatory cavity. It did not look like a lung abscess or carcinoma, but in the region of the hilus there was an indurated area that I thought was carcinoma. The structures about the hilus were almost typical of carcinoma — so much so that when I began to dissect about the hilus, I said, "This is probably an inoperable case; even if I can do a pneumonectomy, it is incurable." There were firm, hard lymph nodes that were beyond the limit of my ability to reach. I completed the pneumonectomy, and I was sure the lung and lymph nodes would show malignant disease. The perihilar tissue — not only the nodes, but also the areolar tissue — was adherent. It was difficult to develop the vessels. There was a good deal of contraction of structures, and the phrenic nerve was pulled over.

Clinical Diagnosis

Bronchiogenic carcinoma of lung?

Dr. Churchill's Diagnosis

Bronchiogenic carcinoma of lung.

Anatomical Diagnoses

Chronic pulmonary tuberculosis.

Tuberculosis of bronchial lymph nodes.

Pathological Discussion

Dr. Sweet: This case was different from the cases of tuberculosis in which we have operated, the majority of which have shown a free hilus, which could be dissected with relative ease. I should like to point out that in the field of thoracic surgery there is an analogy with abdominal surgery in cases of this sort. Often, I have been confronted, as other surgeons have, with the difficulty in differentiating an inflammatory mass in the cecum or sigmoid and a carcinoma. I remember a case that was discussed here of a patient on whom I had operated, performing a right colectomy in the belief that I was dealing with carcinoma. Dr. Castleman came over to see it and could not tell what it was. It turned out to be an appendiceal abscess. Dr. Allen remarked that he had had that experience himself. In other words, on the operating table it is difficult to differentiate some cases of chronic infection or tuberculosis of the colon and carcinoma, and the same thing is true of the lung.

Dr. Mallory: The lung we received showed extensive tuberculosis with a large cavity at the apex of the lower lobe. The lymph nodes at the hilus were also involved with firm, dense, fibrocaseous tuberculosis.

CASE 33052

Presentation of Case

First admission. A forty-eight-year-old machinist was admitted to the hospital because of a productive cough.

At the age of twenty-five the patient had had "double pneumonia" and typhoid fever, and he had suffered from influenza during the 1918 epidemic. At the age of thirty-one a rib resection had been performed because of a left-sided empyema. The patient stated that during the succeeding two weeks he could "blow smoke through a hole in his back"; this closed during the next two and a half months, however, and he fared well except for a chronic dry cough and occasional colds that settled in the "left bronchial tubes." The recent cough had begun nine months previously, when he had had a severe cold progressing to pneumonia with a temperature of 101°F. and a cough productive of "heavy pus" without blood or chest pain. During the two weeks of confining illness, he lost 17 pounds. Following this he continued to have a cough productive of one or two tablespoonfuls of yellow-white, sometimes foul-smelling, sputum, especially in the morning. He also complained of persistent weakness and general malaise. Six months before entry the feet became chilled and the patient was again confined to bed for three days with chills and fever. A similar episode occurred two months later. The patient had never coughed up blood since the original empyema.

There was no family or other history of exposure to tuberculosis. The patient had done considerable welding and had been exposed to zinc oxide.

Physical examination in the Out Patient Department three months before admission revealed that the left leaf of the diaphragm was elevated, with limited excursion; there were a soft inspiratory wheeze over the left base posteriorly and occasional crackling inspiratory rales over the same area with questionable dullness and diminished breath sounds. X-ray examination revealed a cavity filled with air and fluid in the posterior aspect of the left chest at the level of the hilus, where there was a band of increased density reaching to the diaphragm. The left lower lobe appeared to be somewhat decreased in size. In the lower portion of the cavity there was a mottled area of increased density that had the appearance of old lipiodol. There was a partial resection of the ninth rib on the left.

Three months later the patient was admitted to the hospital. The x-ray and physical findings were essentially unchanged. A tuberculin test was negative in a concentration of 1:10,000. Examination of the blood showed a red-cell count of 4,260,000 and a white-cell count 8550, with 40 per cent neutrophils, 49 per cent lymphocytes, 8 per cent monocytes, 1 per cent eosinophils and 2 per cent basophils. Repeated x-ray series were negative.

A right lower lobectomy was performed, and

pathological examination was reported as showing lung abscess with chronic pneumonitis and pleuritis. Following operation there was persistent drainage of 30 to 40 cc. of foul pus from the wound without fever and despite sulfadiazine administration. X-ray examination revealed adequate expansion of the remaining lung, with no mediastinal shift. There were only a small amount of fluid and a few streaks of atelectasis at the base of the lower lobe. Lipiodol injection revealed a fistulous communication with the left lower bronchus with no intervening cavity. The patient was discharged to be followed in the Out Patient Department.

Second admission (four months later). Follow-up study in the Out Patient Department revealed the tract to have gradually closed and after two months extended only into the chest wall. A repeat x-ray examination disclosed essentially the same findings as previously with some thickening of the pleura along the left chest laterally and posteriorly without definite effusion. During the following month the patient had fever and malaise with increased drainage; the fistula reached the pleural space, but no pocket of pus was evident. Two weeks later an abscess developed in the old operative scar. It was incised and drained and found to extend to the bone. Culture revealed a few colonies of *Staphylococcus aureus*.

Two weeks later the patient was readmitted to the hospital because of a new abscess. Incision and drainage of the new area revealed an abundant growth of *Staph. aureus* on culture. The patient was discharged on the seventh day.

Third admission (five weeks later). The drained areas failed to heal, and more furuncles continued to develop so that there were six different areas, most of which drained thin pus with cheesy particles that on smear and culture were negative for tubercle bacilli and showed moderate growth of *Staph. aureus*. The patient was readmitted for biopsy and open drainage of the entire area.

Physical examination showed no changes in the five months since the lobectomy, except for the evident weight loss of 30 pounds and the draining sinuses.

X-ray studies with lipiodol injections of three fistulas were unsatisfactory but revealed no definite interconnection. The white-cell count was 25,000, and the red-cell count was 4,100,000, with a hemoglobin of 65 per cent. Examination of the urine showed only rare, short granular casts and 8 to 10 white cells per high-power field in the sediment.

Drainage of the area was accomplished surgically with a rib resection and secondary closure. Pathological examination of the tissue was reported as showing acute and chronic inflammation, with granulation tissue and a foreign-body, giant-cell reaction. The last x-ray film showed the seventh, eighth and ninth ribs resected posteriorly. The tenth rib was fractured, with a mottled area of decalcification.

Final admission (two months later). The patient was readmitted because of a bronchocutaneous fistula that had developed two weeks previously. He experienced considerable pain and cough during the week before admission and raised small amounts of yellow-white sputum without blood. He complained of profuse night sweats, with a temperature of 101 to 102°F., but there were no chills.

Physical examination was essentially unchanged aside from the emaciated appearance and a new bronchocutaneous fistula.

Examination of the blood revealed a red-cell count of 3,800,000, with a hemoglobin of 80 per cent, and a white-cell count of 21,700. Repeated examinations of the urine were negative. An x-ray film disclosed a small amount of periosteal proliferation of the eighth and ninth ribs and little change in the thickened pleura. Subsequent re-examinations showed a small amount of new-bone formation about the ends of the resected ribs. In addition, there was a moderately large air-containing cavity in the posterior portion of the left lung field with no evidence of undrained fluid.

In the hospital the patient continued to have pain in the ribs on the left, a spiking temperature up to 102.5°F., with profuse night sweats and continued thick yellow-green purulent drainage of the fistulas. On one occasion he coughed up "three mouthfuls" of blood. He was given testosterone and repeated blood transfusions. The white-cell count in two weeks fell to 15,000, and the red-cell count rose to 5,130,000 with a hemoglobin of 85 per cent.

Three weeks after admission an x-ray report mentioned a definite collection of fluid posteriorly in the left chest, probably at the level of the fifth rib. During the following week a new rib resection was performed.

Differential Diagnosis

Dr. F. Dennette Adams: The history at the time of the first admission was characteristic of bronchiectasis. At first glance it seems reasonable to suppose that this patient's trouble started at the age of thirty-one with an attack of lobar pneumonia. Empyema appearing spontaneously is rare, and the spitting of blood was probably due to pneumonia rather than to empyema. The bronchopleural fistula doubtless resulted from rupture of a small subpleural abscess. Following recovery, one might postulate that the process in the lung did not entirely clear; the patient was presumably left with some residual damage, such as an area of bronchiectasis at the site of the original pneumonia. One can assume that, as so often happens in these cases, complicating episodes following the acute upper respiratory infection consisted of repeated bouts of acute bronchopulmonary disease in the affected area.

The physical signs in the thorax — dullness, elevation of the diaphragm, diminished breath sounds, and rales — are consistent with this explanation,

indicating probable pleural thickening and contraction, localized pulmonary fibrosis and bronchiectasis. The localized wheeze is confusing and cannot be accounted for. Usually indicative of partial obstruction of a bronchus, this sign is highly suggestive of tumor. Because of the presence of trapped air, with partial bronchial obstruction, however, one expects expansion, not contraction, of the affected area of the lung; the findings should have been not dullness but hyper-resonance and prolonged expiratory breath sounds. Examination of the lungs provided no evidence of cavitation, probably because of the deep location of the cavity.

May we see the first x-ray films?

Dr. Laurence L. Robbins: These are the first films taken and show the thickened pleura and an air-containing cavity, which appears to be in a dorsal division of the left lower lobe. There is a collapse — that is, a decrease in the size of the lobe — because the lobe is too small generally, but the greater portion of the trouble appears to be in the dorsal division.

Dr. Adams: Could the shadow said to have the appearance of lipiodol be caused by any other foreign substance? We have no record of lipiodol injection.

Dr. Robbins: It could be calcification, I suppose, but it looks much more like lipiodol.

Dr. Adams: Attention must be directed to certain factors in the record that throw considerable doubt on the explanation of the illness just postulated. In the first place the patient had no trouble from the age of thirty-one until nine months before admission, when he was forty-seven and when all the acute episodes occurred; it is hard to believe that with either bronchiectasis or abscess he could have gone sixteen years with no symptoms and then have been repeatedly in trouble. Secondly, at that time, the patient lost 17 pounds within two weeks — a much greater weight loss than is usual in acute infection superimposed on bronchiectasis or in pneumonia. Thirdly, x-ray examination showed an abscess cavity that was thin walled and, although productive of sputum, gave a smaller daily output than is usual in a postpneumonic abscess. These facts force me to the conclusion that the episode nine months before entry was new and not related to the empyema and the postulated lobar pneumonia. The character of the cavity and the amount of sputum are not inconsistent with coccidioidomycosis although typically one sees even less tissue reaction around the cavity than that in this case. This disease can be dismissed on the grounds of probability, since we have no record that the patient ever resided in an endemic area.

The laboratory tests give no clues. The result of the blood Hinton test is not reported.

Dr. Tracy B. Mallory: We can assume that it was negative.

Dr. Adams: All one can say up to the time of the first operation is that the patient had an abscess of undetermined origin, probably of neither tuberculous nor postpneumonic origin.

Following operation the patient was in continuous trouble. At no time was he entirely free of draining sinuses, and after a few months, new abscesses and draining sinuses continued to appear in spite of repeated surgical measures. X-ray study disclosed evidence of bone destruction, and eventually a bronchocutaneous fistula developed. Meanwhile, the patient went downhill, with severe weight loss, increasing fever and marked elevation of the white-cell count. A further indication of his poor condition is the need for transfusions and testosterone, the latter given presumably in an effort to restore nitrogen balance. Sulfonamides and penicillin were doubtless given and proved ineffectual.

Dr. Mallory: The patient received sulfonamides repeatedly. This case occurred before the era of streptomycin. Penicillin was given only toward the end of the last admission.

Dr. Robbins: This film, taken shortly after lobectomy, shows more pleural reaction than one would expect. This next film simply confirms the presence of the bronchocutaneous fistula; the lipiodol enters the left main bronchus and then goes into the portion of the upper lobe. This film was taken approximately five months after the postoperative episode, and there is an increase in the pleural reaction. The changes in the periosteum on the inner surface of the ribs, which usually go with secondary infiltration with empyema, begin to be visible. The resected ribs are again seen, and this film shows beginning new-bone formation, but it does not seem to be so marked as usual. These films show the collection of fluid posteriorly that was mentioned at the time of the final admission. I think that there is some change in this vertebra, but the films were not taken for that purpose.

Dr. Adams: What about the fractured rib?

Dr. Robbins: It does not seem to have regenerated so rapidly as they often do, and in all these films there are questionable areas of bone destruction.

Dr. Adams: Fracture because of destruction?

Dr. Robbins: No; probably the result of operative procedures.

Dr. Adams: We are confronted, then, with a patient who, nine months following an acute pulmonary infection of some kind, was found to have a lung abscess. The clinical picture did not conform to that usually seen in postpneumonic abscess. Following lobectomy, instead of getting well, as was anticipated, the patient continued to be in difficulty. The systemic reaction was severe; multiple abscesses and fistulas and eventually a bronchial fistula developed. The possibility of tuberculosis seems to be ruled out by the x-ray films and the pathological findings, as well as by the negative cultures. I should be more confident if a guinea-pig

test had also been performed and reported negative.

A chronic suppurative process due to *Staph. aureus* is suggested by the repeated isolation of that organism from the pus, but I doubt whether it could be held responsible for the large number of fistulas and, in a chronic case such as this, for the severe systemic reaction. Once drainage had been established, that should have been sufficient. More help might also have been expected from the sulfonamides.

A foreign body, perhaps a sponge, is a possibility, but again one would expect a single and persistent draining sinus — not multiple sinuses.

One not infrequent disease of the lung is manifested by the appearance of multiple stubborn sinuses, and I believe this to be the cause of this patient's illness. Although typically it causes multiple small abscesses of the lung, I suppose it could produce a large one; an explanation is provided for the unusual character of the abscess — that is, for its failure to conform to the pattern of postpneumonic cavitation. This disease also readily accounts for the extreme systemic reaction, loss of weight, emaciation and high fever. In my opinion the only likely diagnosis in this case is actinomycosis.

A PHYSICIAN: Did the yellow pus have any relation to the actinomycosis? Were the sulfur granules cultured?

DR. MALLORY: That is a question that often comes up. In all probability the laboratory was never asked to look for actinomycosis. The specimens were examined in the routine bacteriologic laboratory and repeatedly examined in the surgical bacteriology laboratory, and in neither were actinomyces found; I cannot say how specifically they were looked for, however.

DR. BEVERLY TOWERY: We recently had a patient in the Baker Memorial Hospital with a chronic infection of the chest wall; after two weeks' study we were convinced that she had actinomycosis, and yet it took a week of careful search to find the sulfur granules. I have seen three cases within the year — one with recurring liver abscesses, the case that I just mentioned and another that simulated chronic gall-bladder disease — in which the patients had actinomycosis. It is essential to realize the protean nature of this infection so that its presence may be suspected more frequently and the organisms may be looked for in fresh material.

CLINICAL DIAGNOSIS

Actinomycosis of lung.

DR. ADAMS'S DIAGNOSIS

Actinomycosis of lung, with abscess formation and bronchocutaneous fistula.

ANATOMICAL DIAGNOSIS

Actinomycosis of lung.

PATHOLOGICAL DISCUSSION

DR. MALLORY: At the final operation some tissue was removed in which we were able histologically to demonstrate actinomycosis. We went back over all the previous specimens, including those from the original lobectomy, and in none were we able to find organisms. The organisms are often few and far between. There is a good deal of chance in picking the right location from which to take a section. It is reasonable to assume that the history from the beginning can be explained on the basis of actinomycotic infection.

DR. DONALD S. KING: Are cavities with a fluid level usually seen?

DR. MALLORY: We expect multiple small cavities instead of a single cavity.

DR. ADAMS: I tried to find out about that point from the literature in the short time that I had to prepare this case. Most articles merely refer to cavitation.

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The Journal does not hold itself responsible for statements made by any contributor.

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POSTGRADUATE LECTURE COURSE

The three-month Postgraduate Lecture Course given last winter and spring proved to be so popular that, in a somewhat abbreviated form, it will be repeated this year. The program has been arranged by a subcommittee of the Subcommittee on Postgraduate Education of the Committee on Postwar Planning of the Massachusetts Medical Society, in co-operation with the Massachusetts Department of Public Health. The lectures will be given on Mondays — from 5:30 to 7:00 and from 7:30 to 9:00 — and on Wednesdays — from 3:00 to 6:00 — beginning on March 3 and ending on April 23. A buffet supper will be served on Mondays between 7:00 and 7:30. As formerly, all exercises will be held at Sanders Theater, Harvard University, Cambridge.

A tentative outline of the program appears elsewhere in this issue of the *Journal*. All the topics are of general interest, and the chairmen have been so wisely selected that adequate and timely discussions are guaranteed. Of special interest is the topic for the first day — "The Medical Aspects of Atomic Warfare." The session will be presided over by Dr. Stafford Warren, professor of radiology at the University of Rochester School of Medicine, and Dr. Shields Warren, pathologist at the New England Deaconess Hospital, Boston. The former, while in the Army, served as medical director of the Manhattan Project, and the latter, while in the Navy, was director of medical research at the Bikini Test. They undoubtedly know more about the medical aspects of atomic warfare than any other persons in the world, and the program, which will include motion pictures of the Bikini Test, should provide three hours of intensely interesting instruction and entertainment.

The course is primarily intended for the practicing physicians of Massachusetts and the surrounding states, but hospital residents and interns and medical students are cordially invited to attend. All those who plan to be present are required to register, but no charge will be made other than one for those who participate in the suppers on Mondays. A copy of the final program will be forwarded to all licensed physicians in Massachusetts, and a return postal card will be enclosed requesting information concerning attendance at the first lecture, the majority of lectures and the buffet suppers. To aid the subcommittee, all postal cards should be promptly returned. Those who wish to register immediately should do so by writing to the Postgraduate Lecture Course Committee, Massachusetts Medical Society, 8 Fenway, Boston.

NATIONAL HEART WEEK

Just as modern warfare demands the active cooperation of a large segment of the civilian population, so the war against disease can no longer be left entirely to a relatively small professional body. In girding itself for a vigorous offensive against man's greatest killer the American Heart Association has undertaken a fundamental reorganization involving extensive lay participation. It is seeking not merely

to broaden its membership base but also actually to take nonmedical persons into its executive bodies up to 50 per cent of the membership of each. This means that the lay members may have an equal voice with the physicians in determining the general policies of the association. Such an arrangement cannot fail to be a stimulating one for both groups. At the same time the Scientific Council, open only to members of the American Heart Association who have made significant contributions to knowledge of cardiovascular disease, will maintain the high scientific standards of the association. Another feature of the reorganization is closer integration with the local heart associations, such as the New England Heart Association, including joint membership. Recognizing that rheumatic fever stands out as one of the most urgent unsolved medical problems of the day, the American Heart Association has accepted the responsibility of incorporating the American Council of Rheumatic Fever within its structure. The objectives of the council are to aid and guide local communities in the organization of programs designed to attack the rheumatic-fever problem, to prepare and distribute criteria for the diagnosis of rheumatic fever and rheumatic heart disease, together with recommendations for treatment and convalescent care, to educate groups of physicians, nurses, social workers, teachers and so forth and to sponsor research.

The American Heart Association will carry on the publication of the *American Heart Journal*, *Modern Concepts of Cardiovascular Disease* and special pamphlets. It will provide slides, pictures, exhibits, models and so forth for instruction and will co-ordinate programs of postgraduate education throughout the country among medical schools, hospitals and county societies. It will assist in the development and establishment of standards for accurate diagnosis, clinics, facilities for acute and convalescent hospital care, nursing care, occupational therapy, vocational guidance and medical social service, periodic health examinations and vital statistics. It will carry health education to the public, and it will aid the cardiac patient in securing employment. Finally, it will accept responsibility for stimulating, guiding, co-ordinating, sponsoring and financing research along statistical, clinical, laboratory and epidemiologic lines. The 1947 budget for carrying out this program is $561,000, including $275,000 for research.

The inauguration of the public-education program has been appropriately observed by the proclamation of National Heart Week — February 9 to 15. During this period every medium of information — the press, radio, magazines, motion pictures and special events — will be used to drive home the message that the control of heart disease is the most vital health problem confronting the public today. The campaign will be purely educational, no public drive for funds being contemplated at this time. Physicians will be well advised to be prepared to answer questions from their lay friends and patients regarding the objectives of the American Heart Association.

MASSACHUSETTS MEDICAL SOCIETY

EXECUTIVE COMMITTEE OF THE COUNCIL

The Executive Committee of the Council, in regular session on January 8, 1947, approved the following recommendations submitted by the Committee on Membership:

That the following named fellows, applying for retirement and with all dues paid and in good standing, be allowed to retire under the provisions of Chapter I, Section 5, of the by-laws:

Champion, Merrill E. (Suffolk), 42 Anderson Street, Boston.
Cook, Philip H. (Worcester), 19 Somerset Street, Worcester.
Cummin, John W. (Suffolk), 416 Marlboro Street, Boston.
Hamblet, Mary L. (Essex South), 120 Federal Street, Salem.
Hosley, Walter A. (Middlesex South), Salem Road, R.F.D., Danvers.
Lakeman, Mary R. (Essex South), 108 Federal Street, Salem.
MacDonald, William C. (Middlesex South), 213 Mountain Avenue, Newtonville.
Thayer, Hartley W. (Middlesex South), 355 Walnut Street, Newtonville.
Tucker, S. Chase (Essex South), 379 Lowell Street, Peabody.

That the following named fellows, applying for resignation and with all dues paid and in good standing, be allowed to resign under the provisions of Chapter I, Section 7, of the by-laws:

Burke, Edward W. (Middlesex South), 55 Bradford Boulevard, Tuckahoe, New York.
Pratt, Henry N. (Norfolk), 444 East 68th Street, New York 21, New York.
Spira, Bertram (Barnstable), 626 S. Alvarado Street, Los Angeles, California.
Zielinski, John B. (Bristol South), 781 Carroll Street, Brooklyn, New York.

That the following named fellows, applying for resignation from the Massachusetts Medical Society but in arrears of dues, shall have these dues owed the Massachusetts Medical Society remitted under the provisions of Chapter I, Section 6, of the by-laws and shall then be allowed to resign under the provisions of Chapter I, Section 7, of the by-laws:

Marchand, Jean (Essex South), 210 Lafayette Street, Salem.
Shpiner, Leonard (Middlesex South), 509 Arcade Building, Kankakee, Illinois.
Snow, Robert (Middlesex East), 1146 Gilmer Drive, Salt Lake City, Utah.

That the following named fellow be allowed to change his legal residence from one district society to another, without change of district membership, under the provisions of Chapter III, Section 3, of the by-laws:

Heusner, A. Price (Suffolk), 647 Pleasant Street, East Weymouth.

MICHAEL A. TIGHE, *Secretary*

DEATH

HOWARD — Perez B. Howard, M.D., of Newtonville, died January 18. He was in his seventy-first year.

Dr. Howard received his degree from Harvard Medical School in 1902. He was a fellow of the American Medical Association.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

REVIEW OF COMMUNICABLE DISEASES IN 1946

More than 100,000 cases of communicable disease were reported in Massachusetts during 1946, exceeding the 1945 total by 20,000 cases. The increase was due mostly to the record-breaking number of measles cases that occurred in the spring, complemented by increases in German measles, chicken pox and dog bite. No serious outbreaks of devastating diseases took place during the year, however, and on the whole the experience with communicable disease can be considered as extremely satisfactory.

The Commonwealth was fortunate to escape with only 377 cases of poliomyelitis during an epidemic year that proved to be the greatest the Nation has experienced since 1916, when a total of 1927 cases, with 452 deaths, occurred in Massachusetts. The most alarming development of the past year was the marked increase in diphtheria, which began in July and culminated in a total of 441 cases, more than doubling the incidence for any year since 1937. This strongly indicates the immediate need of intensifying immunization efforts, especially in Boston, Gloucester, Lowell, Lynn, New Bedford, Somerville, Taunton and Worcester, where the disease has been frequent.

The prevalences of other communicable diseases did not show impressive changes during the year. Slight to moderate increases were reported in Salmonella infections, trichinosis, pulmonary tuberculosis and typhoid fever. On the other hand, sharp decreases occurred in the incidences of scarlet fever, mumps and lobar pneumonia. A decline also was reported in bacillary dysentery, meningococcal meningitis and whooping cough.

Lobar pneumonia broke last year's all-time low to reach the lowest incidence in the history of the Commonwealth, with a total of 1484 cases, compared with 2137 in 1945. No cases of smallpox, leprosy or human rabies were reported.

Following is a list of the communicable-disease totals reported in Massachusetts during 1945 and 1946, together with the seven-year medians:

Disease	1946	1945	Seven-Year Median
Actinomycosis	3	2	4
Anterior poliomyelitis	379	527	182
Anthrax	2	4	5
Chancroid	19	20	‡
Chicken pox	11906	10401	11808
Diphtheria	441	194	154
Dog bite	11705	10585	10585
Dysentery, amebic	4	18	3
Dysentery, bacillary	68	245	303
Encephalitis, infectious	8	18	21
German measles	4619	1308	1938
Gonorrhea	5062	5487	4652
Granuloma inguinale	4	4	‡
Hookworm	3	8	0
Infectious diseases of eye:			
Ophthalmia neonatorum	484*	495	675*
Trachoma	2	1	13
Leprosy	0	0	0
Lymphocytic choriomeningitis	4	8	3†
Lymphogranuloma venereum	10	31	‡
Malaria	497	1031	18
Measles	38402	7486	22338
Meningitis, meningococcal	121	161	161
Meningitis, Pfeiffer bacillus	30	32	29
Meningitis, pneumococcal	44	42	52†
Meningitis, staphylococcal, streptococcal and other forms	19	25	26†
Meningitis, undetermined	42	41	74†
Mumps	5811	15727	10491
Pneumonia, lobar	1484	2137	3290
Psittacosis	2	0	0
Rabies (in animals)	2	0	0
Rabies (in human beings)	0	0	0
Rocky Mountain spotted fever	1	1	0
Salmonella infections	179	124	109
Scarlet fever	6080	10373	10373
Septic sore throat	178	200	177
Smallpox	0	0	0
Syphilis	4970	4378	5024
Tetanus	12	12	17
Trichinosis	38	28	28
Tuberculosis, pulmonary	2902	2675	2816
Tuberculosis, other forms	185	164	221
Tuberculosis, hilum	10	14	
Tularemia	2	0	1
Typhoid fever	33	18	50
Typhus fever	3	0	2
Undulant fever	52	49	46
Weil's disease	0	3	2†
Whooping cough	6514	7445	7548

*Includes suppurative conjunctivitis.
†Four-year median.
‡Made reportable December 14, 1943.

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Clinic Consultant
Salem	February 3	Paul W. Hugenberger
Haverhill	February 5	William T. Green
Lowell	February 7	Albert H. Brewster
Greenfield	February 10	Charles L. Sturdevant
Gardner (Worcester subclinic)	February 11	John W. O'Meara
Brockton	February 13	George W. Van Gorder
Springfield	February 18	Garry deN. Hough, Jr.
Pittsfield	February 19	Frank A. Slowick
Worcester	February 21	John W. O'Meara
Fall River	February 24	David S. Grice
Hyannis	February 27	Paul L. Norton

Physicians referring new patients to clinics should get in touch with the district health officer to make appointments.

MISCELLANY

NOTE

The following appointments to the teaching staff of Harvard Medical School were recently announced: Donald Robert Wilson, of Quebec, Canada (B.A. Oxford University, England, 1937, M.D., C.M. University of Alberta 1931-35), research fellow in medicine; Jacques Antoine Carlotti, of Paris, France (M.D. University of Paris 1940), research fellow in medicine; Wade Volwiler, of Athens, Ohio (A.B. Oberlin College 1939, M.D. Harvard University 1943), assistant in medicine; Louis Anthony Sieracki, of Norwood (S.B. University of Indiana 1927, M.D. University of Indiana 1929), instructor in pediatrics; Bertram Selverstone, of Norfolk, Virginia (A.B. Columbia University 1937, M.D. Harvard University 1941), assistant in surgery; Frederic Crosby Bartter, of Boston (A.B. Harvard University 1935, M.D. Harvard University 1940), research fellow in medicine; Thomas Lanier DeLorme, of Randolph (S.B. Howard College, Alabama, 1939, M.D. New York University 1943), research fellow in neurology; Gardner Cowles Quarton, of Cedar Rapids, Iowa (S.B. Harvard University 1940, M.D. Harvard University 1944), assistant in neurology; Gustave Derouaux, of Liége, Belgium (M.D. University of Liége 1938), research fellow in physical chemistry; Constance Elaine Field, of London, England (M.R.C.S., L.R.C.P., B.M., B.S. London 1934, M.D. London 1937, M.R.C.P. 1941), research fellow in pediatrics; Theodore Dunham, Jr., of Pasadena, California (A.B. Harvard University 1921, M.D. Cornell University 1925, A.M. Princeton University 1926, Ph.D. Princeton University 1927), Henry E. Warren Fellow in Surgery; Martin Bernard Williamson, of Las Vegas, New Mexico (S.B. Brooklyn College 1938, A.M. University of Missouri 1940, Ph.D. University of Missouri 1943), instructor in ophthalmic research; Sidney Bennett Luria, of Waterbury, Connecticut (S.B. Middlebury College, 1938, M.D. Harvard University 1943), assistant in surgery; Ralph Henderson Kellogg, of Saratoga Springs, New York (A.B. University of Rochester 1940, M.D. University of Rochester 1943), teaching fellow in physiology; Somers Hayes Sturgis, of Cambridge (A.B. Harvard University 1927, M.D. Harvard University 1931), assistant in gynecology; Arthur Stanwood Pier, Jr., of Milton (A.B. Harvard University 1935, M.D. Harvard University 1939), assistant in medicine; Beverly Todd Towery, of Bowling Green, Kentucky (S.B. Western State Teachers College, 1936, M.D. Vanderbilt University 1940), research fellow in medicine; and John Bruton Stanbury of Mendota, Illinois (A.B. Duke University 1935, M.D. Harvard University 1939), research fellow in pharmacology.

The appointment of Paul Hathaway Keyes, of Long Island, New York (D.D.S. University of Pennsylvania 1941, A.B. University of Rochester 1945, M.S. University of Rochester 1947), as research fellow in dental medicine, was also announced recently.

CORRESPONDENCE

TRYPTOPHANE AND BLOOD SUGAR LEVELS

To the Editor: As an incident to the experimental ingestion of crystalline *d-l*-tryptophane, it was observed that on ingestion of this essential amino acid, there was, for the persons concerned, a slight but consistent drop in the blood sugar level. Before beginning ingestions on a large scale in an attempt to decrease the caries rate, a small group of young adults volunteered to undergo an experiment to test the blood sugar drop. A dose of tryptophane varying from 0.14 to 0.32 gm., being adjusted to body weight, was ingested by these subjects on four successive days. Then, after a day's rest, the same dose of trytophane was ingested on four more successive days. The material was taken midway between breakfast and lunch, in crystalline powder form, with as much water as desired. Previous ingestions in gelatin capsules had proved less effective in changing the salivary amylase. All blood samples were taken after a five-hour fast, and the last dose of tryptophane was administered two hours prior to drawing the second samples.

The changes in fasting blood sugar levels following these ingestions are listed below and appear to be significant.

SUBJECT	BLOOD SUGAR BEFORE TRYPTOPHANE *mg./100 cc.*	BLOOD SUGAR AFTER TRYPTOPHANE *mg./100 cc.*
R. C.	91	72
G. M.	75	67
P. H.	87	89
N. T.	113	63
L. W.	87	71
B. V.	96	64
S. T.	106	88
N. B.	101	66
O. F.	101	67

Among the nine subjects, only one failed to show a decrease in the blood sugar level, and in this instance the level increased from 87 to 89 mg. per 100 cc. For the other eight, the average fasting blood sugar was 96 mg. (range, 75 to 113 mg.) per 100 cc., and that after the tryptophane was 70 mg. (range, 63 to 88 mg.). This average drop of 26 mg. may be of interest in diabetes and possibly in hypoglycemia. In two control subjects, from whom blood samples were obtained at the same time as those from the test subjects, there was an average gain in the blood sugar level of 3 mg. per 100 cc.

NAOMI COCKE TURNER
GEORGE EDWARD CROWELL

Forsyth Dental Infirmary
140 Fenway
Boston 15

BOOK REVIEWS

Pulmonary Edema and Inflammation: An analysis of processes involved in the formation and removal of pulmonary transudates and exudates. By Cecil K. Drinker, M.D., D.Sc. 8°, cloth, 106 pp., with 27 illustrations. Cambridge: Harvard University Press, 1945. $2.50.

This monograph is divided into five chapters. The first, a discussion of the anatomy of the lungs as related to the problem of transudation, includes a great deal of information that is of clinical significance — notably, a reminder that the lung capillary endothelium depends for its oxygen on the air reaching it rather than on arterial blood and that exudates or transudates in this area must largely be removed through the bottleneck of the lymphatic ducts.

The second chapter discusses the balance of forces that regulate the movement of fluids through the walls of capillaries. A contrast is made between the capillary situation within the lungs and elsewhere in the body, and evidence is presented that the lung is a relatively ineffective organ for ridding itself of excessive transudation; besides anatomic peculiarities, it suffers the handicap of contending against a variable degree of negative pressure within the thorax that acts toward drawing fluid from the pulmonary blood stream.

The third and fourth chapters discuss the treatment of pulmonary edema. There appear to be two appropriate lines of attack: the combating of anoxia and resistance to the tendency of the negative pressure normally present within the chest to increase the flow of water from the lung capillaries. For the former the proper use of oxygen appears clearly indicated; for the latter, no particularly good method is at present available.

The final chapter is concerned with artificial respiration. The author believes that too little attention has been paid to the possibly harmful effect on the lungs of methods usually employed, and he predicts future modifications that will be valuable.

This book contains much more than an original discussion of applied physiology. It is prepared by a talented author who is also an artistic teacher; each chapter is readable, demonstrates on almost every page both scholarship and the writer's familiarity with every stone of the structure on which he has built his views. On the whole, the work deserves high praise; it is not only valuable for reference but also a delightful model for those interested in the art of medical writing.

American Pharmacy: *Fundamental principles and practices, pharmaceutical preparations, biologicals*. Editor-in-chief, Rufus A. Lyman, M.D. 4°, cloth, 540 pp., with 197 illustrations. Philadelphia: J. B. Lippincott Company, 1945. $8.00.

Dr. Lyman and his group of twenty-two collaborators have produced an up-to-date authoritative treatise covering the whole field of pharmacy in a comprehensive manner. The book represents a new trend in textbook construction in pharmacy and has been designed as a teaching textbook and to serve the practicing pharmacist as a working tool. The authors are specialists in their various fields. The work is divided into three parts. The first part deals with the fundamental principles of the basic sciences as applied to pharmaceutical processes, the second covers the field of pharmaceutical preparations, with emphasis on the processes involved in their manufacture, and the third discusses products of biologic origin (the vitamins, hormones and endocrine preparations). The text is well written and printed in two columns with a good type on good paper. The book is recommended for all medical libraries and should prove valuable as a reference source for all public libraries.

The Way of an Investigator: *A scientist's experiences in medical research*. By Walter B. Cannon, M.D. 8°, cloth, 229 pp. New York: W. W. Norton and Company, Incorporated, 1945. $3.00.

The late Dr. Cannon, in this delightful semiautobiography, based on more than forty years of his life spent in the Harvard Medical School, expounds the philosophy of the scientific investigator. He discusses the qualifications of the ideal scientific worker, the necessity of perseverance and stamina, the part intuition and accident play in discovery and the problems that arise from the possible errors and misjudgments that are bound to occur in research. Professor Cannon tells of his early home and college life, his travels abroad, his war experiences, his vacations and his years as professor of physiology. The text is written in an easy, interesting style, and this book is worthy of a place in all libraries, medical, scientific and general, and in all private medical collections.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Plaster-of-Paris Technique in the Treatment of Fractures and Other Injuries. By Lieutenant Colonel T. B. Quigley, Medical Corps, Army of the United States, instructor in surgery (on leave of absence), Harvard Medical School, and junior associate in surgery (on leave of absence), Peter Bent Brigham Hospital, Boston. 8°, cloth, 107 pp., with 103 illustrations. New York: The Macmillan Company, 1945. $3.50.

This manual considers only the mechanical application with one method of treatment of traumatic injuries and only brief mention of the common indications for each cast is made. The diagnosis, evaluation, pathology and prognosis of trauma are not considered. The majority of casts used in ordinary practice are described.

Hematology. By Willis M. Fowler, M.D., professor of internal medicine, University of Iowa. With a chapter by Elmer L. Degowin, M.D., assistant professor of internal medicine, University of Iowa. 8°, cloth, 409 pp., with 110 illustrations. New York: Paul B. Hoeber, Incorporated, 1945. $8.00.

This new textbook on hematology, which was developed by the author from his lecture notes covering the period of the last twelve years, was written primarily for the medical student and the general practitioner, not being designed to serve as a general reference work for those specializing in hematology. The author maintains the viewpoint that hematology is a part of internal medicine rather than a specialty in itself and presents the subject in this light, stressing the clinical and therapeutic aspects of the various diseases. The book is well printed on good paper, with a good type, but is rather heavy for its size.

Clinical Atlas of Blood Diseases. By A. Piney, M.D., M.R.C.P., physician, St. Mary's Hospital for Women and Children, London; and Stanley Wyard, M.D., F.R.C.P., physician, Royal Cancer Hospital, and Princess Beatrice Hospital, London. Sixth edition. 8°, cloth, 137 pp., with 48 illustrations. Philadelphia: The Blakiston Company, 1945. $5.00.

The value of this small atlas is demonstrated by the need of six editions during a period of fifteen years. The book is primarily an atlas, but condensed descriptions of blood conditions accompany the plates.

Amputation Prosthesis: *Anatomic and physiologic considerations, with principles of alignment and fitting designed for the surgeon and limb manufacturer*. By Atha Thomas, M.D., associate professor of surgery (orthopedics), University of Colorado School of Medicine; and Chester C. Haddan, president, Association of Limb Manufacturers of America. 8°, cloth, 350 pp., with 207 illustrations. Philadelphia: J. B. Lippincott Company, 1945. $8.00.

In this monograph, written jointly by a surgeon and a manufacturer of artificial limbs, the problems of the patient, the surgeon and the appliance maker are discussed on common ground and emphasis is placed on the most satisfactory end results for the patient. The importance of anatomic and physiologic considerations in doing an amputation is stressed in relation to established principles of alignment and fitting. The book is designed as a practical guide for the proper use of prosthetic appliances. This unusual book should be in all medical libraries.

Structure and Function of the Human Body. By Ralph N. Baillif, Ph.D., assistant professor of anatomy, Louisiana State University School of Medicine, New Orleans; and Donald L. Kimmel, Ph.D., associate professor of anatomy, Temple University School of Medicine, Philadelphia. 8°, cloth, 328 pp., with 158 illustrations. Philadelphia: J. B. Lippincott Company, 1945. $3.00.

The objective of this new manual is to provide a carefully organized text emphasizing the relation of structure and function. The manual, which is written primarily for students, emphasizes the unity of structure and function. The material is well organized and well printed, with a good type, and should prove valuable to all those who have need of such a manual. A special glossary is appended to the text.

Analyze Yourself: *Enabling anyone to become deeply psychoanalyzed without a personal analyst*. By E. Pickworth Farrow, M.A., D.Sc. With a foreword by the late Professor Sigmund Freud, M.D., LL.D. New York: International Universities Press, 1945. $2.00.

This small book contains a full and detailed account of how to follow a practical method of psychoanalysis of one's own mind. The author used the method on himself and relates his own experiences in recapturing his memory extending over a period of years.

An Introduction to Physical Anthropology. By M. F. Ashley Montagu, B.S., Ph.D., associate professor of anatomy, Hahnemann Medical College and Hospital, Philadelphia, and visiting lecturer, Department of Sociology, Harvard University. 8°, cloth, 325 pp., with 25 illustrations. Springfield, Illinois: Charles C Thomas, 1945. $4.00.

This manual has been written for the general reader and the student, and it is hoped that it will prove of value to biologists, physicians and psychologists. The author has endeavored to present the fundamental facts and problems necessary for the understanding of man. He discusses in order the primates as a zoological group, the origin and evolution of the primates and of man, the criteria and mechanism of ethnic group differentiation, the divisions and ethnic of groups of man, the relation between body, mind and culture, heredity and the influence that the environment has on man. An appendix comprises a practical synopsis of methods of measurement in physical anthropology. In addition to lists of references for further reading appended to each chapter, a general bibliography concludes the volume. The text is easy to read, and the book is printed with a good large type, on good paper. This introduction should prove valuable to all those desiring a short text on the subject.

NOTICES

ANNOUNCEMENTS

Dr. Archie A. Abrams calls attention to the omission of his office address and telephone number from the current telephone directory. His office is at 1093 Beacon Street, Brookline, and the telephone number is ASPinwall 8951.

Dr. Volta R. Hall announces the opening of his office for the practice of psychiatry at 372 Marlborough Street, Boston.

Dr. Merrill Moore has returned from military service and announces the opening of his office at 433 Marlborough Street, Boston. His telephone number is COMmonwealth 4216.

Dr. Bernard J. Niemiro, of 207 Elm Street, Holyoke, announces that in the future his practice will be limited to proctology.

Dr. Robert R. Shapiro announces the removal of his office for the practice of general surgery and of surgery of the sympathetic nervous system from 319 Longwood Avenue to 31 Bay State Road, Boston.

JOSEPH H. PRATT DIAGNOSTIC HOSPITAL

Bennet Street, Boston
Lecture Hall, 9-10 a.m.

MEDICAL CONFERENCE PROGRAM

Wednesday, February 5 — The Use of Isopropyl-aminoethanol in the Management of Bronchial Asthma. Dr. Maurice S. Segal.
Friday, February 7 — Diseases of the Veins in Older Patients. Dr. Edward A. Edwards.
Wednesday, February 12 — Incomplete and Complete Lacerations of the Perineum: Their surgical management. Dr. Louis E. Phaneuf.
Friday, February 14 — Clinicopathological Conference. Drs. Chester S. Keefer and H. E. MacMahon.
Wednesday, February 19 — Pediatric Clinicopathological Conference. Drs. James M. Baty and H. E. MacMahon.
Friday, February 21 — The Mechanism of Hemoglobinuria in Thermal Burns. Dr. Thomas Hale Ham.
Wednesday, February 26 — The Use of Streptomycin in Tuberculosis. Dr. Lowrey F. Davenport.
Friday, February 28 — Intracranial Aneurysms. Dr. William H. Sweet.

On Tuesday and Thursday mornings, Dr. S. J. Thannhauser will give medical clinics on hospital cases. On Saturday mornings, clinics will be given by Dr. William Dameshek. Medical rounds are conducted each weekday by members of the staff from 12:00 to 1:00 in the Lecture Hall.

All exercises are open to the medical profession.

NEW ENGLAND DERMATOLOGICAL SOCIETY

The regular meeting of the New England Dermatological Society will be held in the Skin Out Patient Department of the Massachusetts General Hospital on Wednesday, February 12, at 2:00 p.m.

TUFTS ALPHA OMEGA ALPHA

The Tufts chapter of the Alpha Omega Alpha will meet in the auditorium of the Beth Israel Hospital, Boston, on Wednesday, February 5, at 8:30 p.m. Dr. Erich Lindemann will speak on the subject "Some Psychologic Problems in Medicine."

APPOINTMENT OF U. S. P. H. S. MEDICAL OFFICERS

A competitive examination for appointment in the Regular Corps in the grades of assistant surgeon (first lieutenant) and senior assistant surgeon (captain) will be held in the near future.

Regular Corps appointments are permanent in nature and provide opportunities to qualified doctors for a life career in one or more of a large number of fields, including research, general hospitals, special hospitals, foreign duty and federal, state and local public-health programs. Assignments are made with all possible consideration of the officer's demonstrated abilities and experience. There is ample opportunity for professional growth and development.

All applicants must be at least twenty-one years of age, must be citizens of the United States, must present a diploma of graduation from a recognized medical school and must satisfactorily pass a physical examination performed by Public Health Service officers when directed upon successful completion of the oral and written examination.

Applicants for the grade of assistant surgeon must have had at least seven years of educational and professional training or experience exclusive of high school. Applicants for the grade of senior assistant surgeon must have had at least eleven years of educational and professional training or experience exclusive of high school.

Application forms may be obtained by writing to the Surgeon General, United States Public Health Service, Washington 25, D. C. These should be retained and presented to the Board at the time the applicant appears for the oral examination.

Entrance pay for assistant surgeon with dependents is $3811 a year, and for senior assistant surgeon with dependents is $4351 a year. Promotions are at regular intervals up to and including the grade of medical director, which corresponds to full colonel at $8551 a year. Retirement pay at sixty-four is $4950 a year. Full medical care, including disability retirement at three-fourths pay, is provided. All expenses of official travel are paid by the Government. Thirty days' annual leave with pay is provided.

Examinations will be oral and written. The oral examination will be held at several places throughout the country; in the New England area it will start at 10:00 a.m. on March 4 at the Marine Hospital, 77 Warren Street, Brighton, Boston. The written examination will be held at 9:00 a.m. on March 27 and 28 at places convenient to the candidate and the Service. National Board grades may be used in place of the written examination for the grade of assistant surgeon.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, FEBRUARY 6

FRIDAY, FEBRUARY 7

*9:00–10:00 a.m. Diseases of the Veins in Older Patients. Dr. Edward A. Edwards. Joseph H. Pratt Diagnostic Hospital.
*10:00 a.m.–12:00 m. Medical Staff Rounds. Peter Bent Brigham Hospital.

MONDAY, FEBRUARY 10

*12:15–1:15 p.m. Clinicopathological Conference. Peter Bent Brigham Hospital.

TUESDAY, FEBRUARY 11

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.
*8:00 p.m. Harvard Medical Society. Amphitheater, Peter Bent Brigham Hospital.

WEDNESDAY, FEBRUARY 12

*9:00–10:00 a.m. Incomplete and Complete Lacerations of the Perineum: Their surgical management. Dr. Louis E. Phaneuf. Joseph H. Pratt Diagnostic Hospital.
*11:00 a.m.–12:00 m. Medical Clinic. Amphitheater, Children's Hospital.
*12:00 m. Clinicopathological Conference. (Children's Hospital.) Amphitheater, Peter Bent Brigham Hospital.
2:00 p.m. New England Dermatological Society. Skin Out Patient Department, Massachusetts General Hospital.
*2:00–3:00 p.m. Combined Clinic by the Medical, Surgical and Orthopedic Services. Amphitheater, Children's Hospital.
*7:15 p.m. Graduate Seminar in Pediatrics. Children's Medical Service, Amphitheater 3A, Massachusetts General Hospital.

*Open to the medical profession.

(*Notices continued on page xiii*)

NOTICES (*Continued from page 190*)

OCTOBER 11–MAY 14. Metropolitan State Hospital. Page 398, issue of September 12.

FEBRUARY 4. Greater Boston Medical Society. Page 156, issue of January 23.

FEBRUARY 5. Tufts Alpha Omega Alpha. Page 190.

FEBRUARY 5–28. Joseph H. Pratt Diagnostic Hospital. Medical Conference Program. Page 190.

FEBRUARY 7. American Board of Obstetrics and Gynecology. Page 44, issue of January 2.

FEBRUARY 9. National Conference on Medical Service. Page 956, issue of December 26.

FEBRUARY 12. Massachusetts Medico-Legal Society. Page 156, issue of January 23.

FEBRUARY 12. New England Dermatological Society Page 190.

FEBRUARY 13. The Clinical Importance of the Blood Type in Transfusions and in Obstetric Patients. Dr. Louis K. Diamond. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

FEBRUARY 26. Tufts Alpha Omega Alpha. Page 156, issue of January 23.

MARCH 13. Cornell Medical Alumni Day. Page 156, issue of January 23.

APRIL 28–MAY 2. American College of Physicians. Page 206, issue of August 8.

MAY 20–22. Massachusetts Medical Society. Annual meeting. Hotel Statler, Boston.

JUNE 9–13. American Medical Association. Page 698, issue of November 7.

JUNE 30–JULY 3. American Urological Association. Hotel Statler, Buffalo.

SEPTEMBER 8–12. American Congress on Obstetrics and Gynecology. Page 636, issue of October 24.

DISTRICT MEDICAL SOCIETY.

PLYMOUTH

FEBRUARY 20. Moore Hospital, Brockton.
MARCH 20. Goddard Hospital, Brockton.
APRIL 17. State Farm, Bridgewater.
MAY 15. Lakeville Sanatorium, Lakeville.

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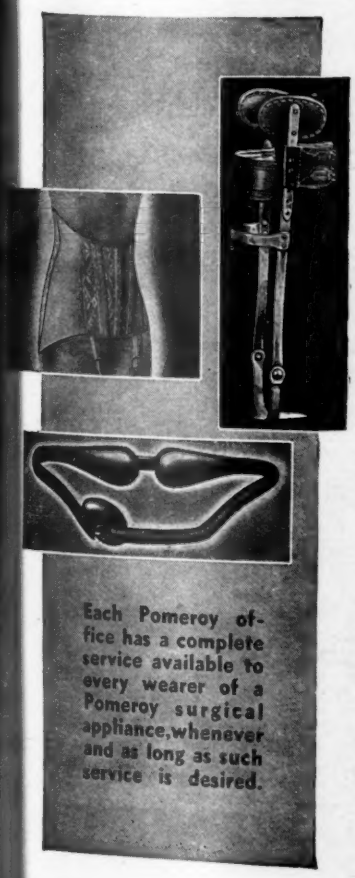
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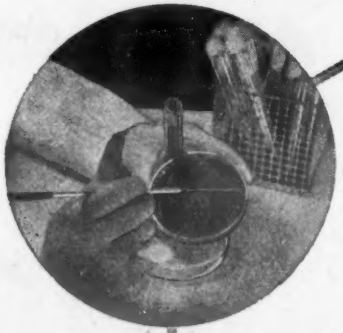

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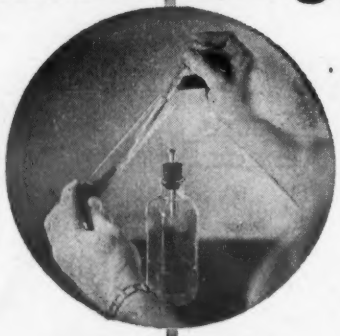

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